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SPECIAL NOTICE

Beginning with the October 1950 issue, it is planned to introduce several changes in the covers of all the A. M. A. specialty journals.

The cover of this journal will be white enamel stock with title identification as the A. M. A. Archives of Otolaryngology in color, with date, volume and number also in color. The Table of Contents, which is now carried on the back cover, will be placed in the front of the journal directly preceding the lead article or editorial. Second, third and fourth covers will be used for color advertising.

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Other factors which make it difficult for the hard-of-hearing to obtain employment are the various compensation laws. Designed to protect the rights of workers, they militate against those suffering from physical handicaps. Employers often have the fear—unsubstantiated as it is—that the hard-of-hearing are more prone to accidents than those with normal hearing.[1] There is also a belief that certain occupations are hazardous for those with hearing defects.[1]

When employment is at a high level, the hard-of-hearing may experience little difficulty in finding or holding positions. It would be unrealistic to deny, however, that when competition is high, that "when a reduction . . . is called for in the labor force of some industrial undertaking, it may at times be the deaf who are first to suffer. With an oversupply of labor it is they who feel the effects most keenly. Upon them times of depression bear disproportionately hard." [1]

A HEARING DEFECT NEED NOT BE AN ECONOMIC HANDICAP

Though deafness has been called "a very serious national menace interfering with efficient work and with the financial return for such work," [2] it does not follow that those with hearing defects cannot be gainfully employed. Though the patient with increasing acoustic difficulties may feel discouraged at the prospect of keeping his place in a competitive world, he should and can be made to see that "he can be equipped to meet the competitions with new abilities and skills. The patient," McIntire [3] emphasizes, "must be made aware that he is not a deafened man who must find ways to appear normal, but rather that he is a relatively normal man and must find ways to show that he is a successful deafened man."

And, in truth, the hard-of-hearing "can and do hold their own in the economic world." [4] Of course those with hearing defects cannot hope to work in professions or vocations where acute hearing is necessary, but outside of such limited fields their defects need be of little importance if they make use of the assistance available to them.[4] "Where the individual successfully avails himself of the adjustments or crutches supplied by lip reading and the electrical hearing aid, his economic value remains," Berry [4] asserts.

Of course, as Berry [4] points out, the worker with defective hearing needs to compensate in other ways for his handicap: "He may be neater, more prompt, more solicitious; he may spend less time in fooling while at work . . ." In short, he must do everything to make himself a more valuable employee than his competitors.

The physician, of course, has an important role in handling the economic life of his hard-of-hearing patient. He "may fittingly consider the vocational aspect as being part of his responsibility." [4]

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We have progressed far from the days of Aristotle when a deaf child was believed less useful than an animal.[5] Today he is "a vital factor in the economics of any country." [5]

It is early in childhood that the vocational problem presents itself and "the issue must be met early." [4] The handicap recognized, the outlook is good. "A partial hearing impairment, incurred after good speech and an average education have been acquired, does not present any great occupational problem." [4]

With the recognition that a child has a hearing defect, his course in life should be laid out with a common sense consideration that some professions and vocations are closed to him. He can, for instance, scarcely be encouraged to become either a violin virtuoso or a piano tuner. This does not mean, however, that he cannot be fitted to earn a good living in many other fields of endeavor. And with a mechanical aid to augment his hearing, and the encouragement to make full use of it, his economic future can certainly be made secure.

1. Best, Harry: Deafness and the Deaf in the United States. The Macmillan Co., New York, 1943, Chap. 16.
2. Barnett, H. N.: M. Press **216**: 84-85 (July 31) 1946.
3. McIntire, Ross T.: Postgrad. Med. **1**: 175 (March) 1947.
4. Berry, G.: In Nelson Loose-Leaf Medicine of the Ear. Edit. by E. P. Fowler, Jr., Thomas Nelson and Sons, New York. 2nd ed. 1947, Chap. 16.
5. Martin, G. E.: Edinburgh, M. J. **54**: 158-166 (March) 1947.

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[1]Cooke, R. A.: *Allergy in Theory and Practice*, Philadelphia: W. B. Saunders Company, 1947, p. 186

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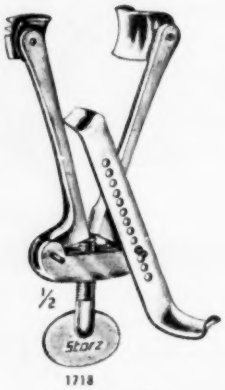

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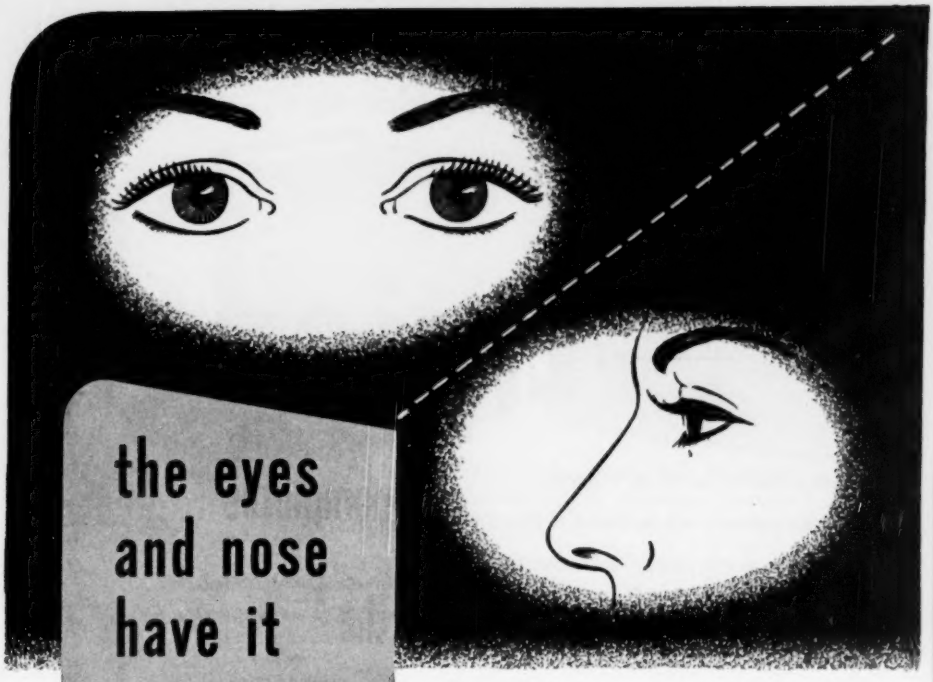

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1. Waldbott, G. L.: Management of Allergic Eczema. Am. Pract. 4:59-63 (Oct.), 1949.

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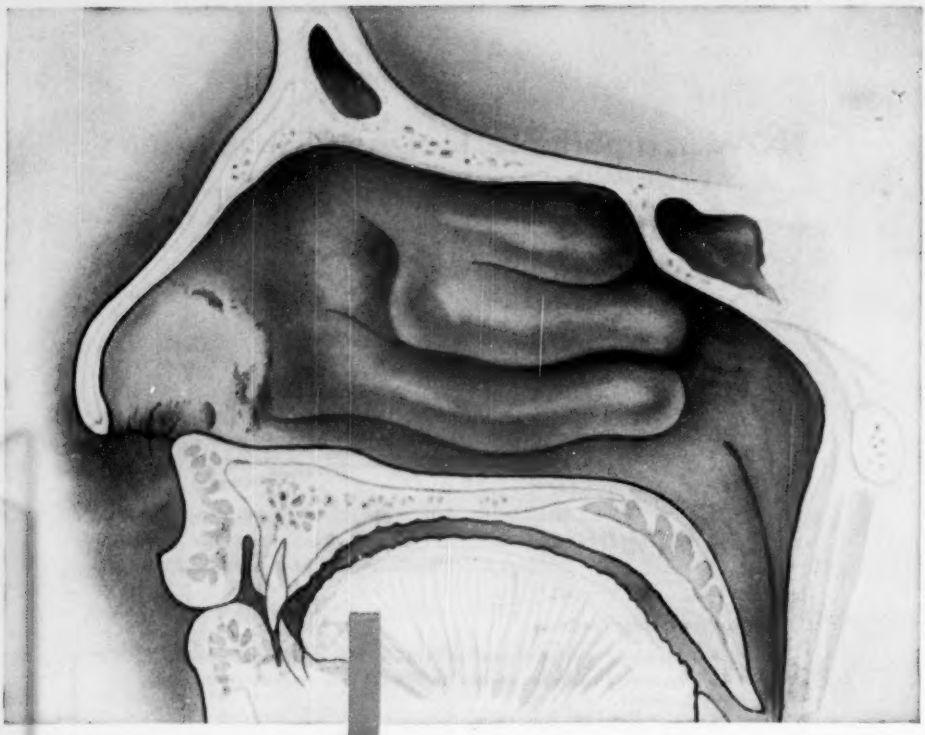

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MECHANISM OF SUFFOCATION IN SPINOBULBAR POLIOMYELITIS AND EXPERIENCES WITH OPERATIVE TREATMENT

ARNE SJÖBERG, M.D.
STOCKHOLM, SWEDEN

DURING the last five years a wave of enthusiasm for tracheotomy in bulbar poliomyelitis has swept over the United States. At the First International Poliomyelitis Conference,[1] held in New York from July 12 to 17, 1948, it was generally agreed that in certain cases tracheotomy must undoubtedly be considered as directly lifesaving.

Already in 1931 Wilson recommended tracheotomy and cited a few successful cases. Well over a decade afterward the idea was taken up anew, and in 1943 Galloway [2] published reports on 3 tracheotomies, with 1 death. In 1946 a great epidemic raged in Minneapolis. Diagnosis was made in about 400 bulbar cases, and Priest, Boies and Goltz [3] reported 75 cases of tracheotomy with survival in 29.

In 1948 Davison [4] reported 15 cases without fatal issue, and in September 1949 Galloway also reported 15 cases, likewise without fatal issue.

In Stockholm the problem became acute in an almost frightening and dramatic way during the autumn of 1949, when patients with severe cases of bulbar poliomyelitis literally streamed into the Hospital for Infectious Diseases within the space of a few weeks.

The situation is quite new for the North, and the question will probably have great importance for epidemiologists, but intensely active

From the Hospital for Infectious Diseases, Otolaryngological Department (Chief, Dr. Arne Sjöberg).

This article is founded on two lectures, one given at the annual meeting of the Swedish Association of Epidemiologists, held at the Hospital for Infectious Diseases, Nov. 24, 1949, and one at the annual meeting of the Swedish Oto-Laryngological Association, held at the Eastman Institute, Nov. 26, 1949, Stockholm.

1. Proceedings of the First International Poliomyelitis Conference, Philadelphia, J. B. Lippincott Company, 1949.

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3. Priest, R. E.; Boies, L. R., and Goltz, N. F.: Tracheotomy in Bulbar Poliomyelitis, Ann. Otol., Rhin. & Laryng. **56**:250 (June) 1947.

4. Davison, F. W.: Acute Laryngotracheobronchitis: Further Studies on Treatment, Arch. Otolaryng. **47**:455 (April) 1948.

teamwork is required also on the part of otologists attached as consultants to the hospitals for infectious diseases.

Meanwhile, the difficulties are so numerous and varied that it is impossible at this early stage to foresee the final results.

Everyone knows how poliomyelitis varies in its details from patient to patient, thus rendering statistical analysis of the results of the treatment comparatively uncertain.

In order to collect control material and thus give an idea of the extent of the problem in Sweden, I shall briefly cite from an investigation carried out by Bergman and Huldt [5] on the results of respirator treatment in poliomyelitis.

In 1934 Sahlin respirators were installed, and in 1948 about seventy of these were in use in the country. The Royal Medical Board is of the opinion that there ought to be one respirator per 100,000 inhabitants.

During the twelve years from 1934 through 1945, 158 patients were treated at the Hospital for Infectious Diseases in Stockholm; 61 per cent of these were men. Only 28 patients are definitely known to have survived, a rate of 18 per cent. The high mortality rate of 82 per cent is much higher than the rate in cases of paresis in general, in which it varies between 10 and 20 per cent.

Bergman and Huldt presented an account of 834 cases of poliomyelitis for the whole country, in which 63 per cent of the patients were men, a fact in agreement with earlier observations that poliomyelitis affects men oftener than women.

The mortality rate for the whole of Sweden is extremely high, 86 per cent (119 survived). Bergman and Huldt stated that the causes of death were pareses of the respiratory muscles or circulatory collapse.

By virtue of the respirator treatment, the life of every seventh patient was saved; 3 out of 100 were able to work and lead a socially useful life; 5 out of 100 became only partially capable of working, and 4 to 5 per cent remained total invalids.

In principle, poliomyelitis can be fatal in three different ways.

1. The commonest fatal form is the peripheral respiratory paresis, in which the respiratory musculature, the intercostal muscles and the diaphragm are paralyzed. By protracted artificial breathing in the respirator, the patient quickly adjusts himself to its rhythm, and in most of these cases life can now be saved, thanks to the prophylactic antibiotic treatment which prevents pulmonary complications.

2. In the graver, more unusual cases, localization of the virus lesion is exclusively bulbopontine. Earlier, the cause of death was considered to be paralysis of the circulatory and respiratory centers. In this

5. Bergman, R., and Huldt, S.: The Swedish Results of Respirator Treatment in Poliomyelitis, Nord. hyg. tidskr., 1948, no. 5, p. 135.

connection, I should like to point out that earlier most of the research workers on poliomyelitis seemed to agree that in many cases it is difficult or impossible to determine the cause of death with clinical certainty.

3. If the virus lesions are disseminated there ensues lethal bulbospinal combined lesions.

The symptomatology of bulbar poliomyelitis is dominated by lesions of the nuclear cranial nerves, of which 90 to 96 per cent consist of dangerous paralyses of the vagus. After a critical analysis of the varying picture of symptoms of bulbar poliomyelitis, it is now agreed in the United States that the alterations lead to hypoxia, with its well known consequences.

In the United States this opinion seems to have been generally accepted, and preventing obstruction of the air channels during the treatment is considered essential. The war cry is now "Prevent anoxia," and efforts are directed toward preventing the accumulation of carbon dioxide by early tracheotomy, toward postural drainage with suction, control of blood pressure, endoscopic bronchial toilet and the administration of oxygen.

Meanwhile, it is remarkable that the American investigators, especially Galloway, maintain that secretory aspiration, due to paralysis of the pharynx and its consequent deglutitive inability, is the principal cause of death by suffocation.

At the Hospital for Infectious Diseases there have been 14 patients with this condition under treatment with only 2 fatal issues. The majority of the patients were men; only 2 were women. The youngest patient was 5½ years old; the oldest was 40. These cases happened to be so representative and together formed such a complete chart of the different types of poliomyelitis which lead to death by suffocation that I can use them as a frame for a schema of the mechanism of suffocation, including the indication for tracheotomy. The cases can thus be divided into five groups.

Group 1.—*Peripheral Respiratory Paresis Without Simultaneous Paresis of the Pharynx.*—The symptoms of suffocation occur when the patient aspirates secretion from the bronchial tree and when, during respirator treatment, saliva is likewise sucked down into the tracheobronchial tree. Frequently repeated bronchoscopic suction, with or without preceding tracheotomy, saves the perilous situation.

Group 2.—*Suffocation Due to Aspiration in Clearly Bulbar Cranial Nerve Lesions, Resulting in Bilateral Paresis of the Pharynx.*—The most dramatic of the cases is a good illustration.

A 39 year old man, strong and previously quite healthy, showed a complete bilateral paresis of the pharynx with inability to swallow and normal movements of the vocal cords. There were no spinal lesions nor peripheral respiratory paresis.

The "gurgling" sound in breathing was significant, being caused by a beginning accumulation of frothy saliva in the buccal cavity between the lips and, lower down, in the orifice of the hypopharynx. The saliva showed a tendency to run partly down into the larynx lumen along the arytenoid regions. Nourishment was given by a feeding tube passed through the nose. The patient was very restless and was given oxygen at intervals, but he could breathe freely. Suddenly, one morning, he was found, to all appearances, lifeless and blue; he was not breathing, but the heart was beating and the pulse was palpable. He was hastily placed in the respirator, but, of course, in vain. I was called immediately and succeeded in

Fig. 1.—A doubly vagotomized and tracheotomized cat, with contracture of the upper ostium (*A*) when the head is held downward.

inserting a bronchoscope into the trachea. Gall-colored ventricle content literally spurted out of the bronchoscope, forming a pool on the floor. Immediately after this I did a tracheotomy toward the bronchoscope. The respirator was started anew, and out of the tracheostoma now spurted a grayish white gruel-like substance to a height of at least 1 decimeter above the wound. After this procedure the bronchial tree was carefully sucked clean, and penicillin was then instilled. Breathing reestablished itself quickly, and the patient recovered with astonishing rapidity. From then on he did not need the respirator, being able to breathe freely, and on the fifth to the sixth day he could sit up in bed and stand up. He was decannulated after five weeks. The pharyngeal paresis was compen-

sated after six to seven weeks, and after four months he was quite fit to work. He did not have any spinal pareses.

In this case there occurred an aspiration of saliva plus vomitus. This case taught me that tubal feeding can be dangerous in the early stages. Therefore, this patient was subsequently given, to begin with, fluid rectally, aminosol* (modified fibrin hydrolysate) intravenously and saline solution parenterally.

For the following bronchial infection, he was given, prophylactically, penicillin in large doses, aureomycin and, for a short period, also dihydrostreptomycin.

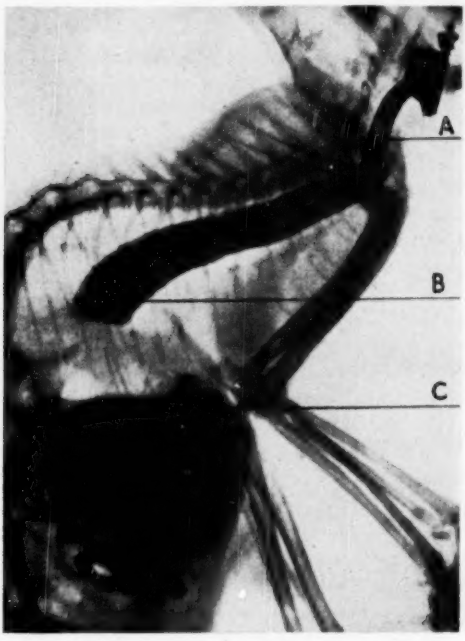

Fig. 2.—Contracture of the cardia when the head is held up. *A* indicates the tracheal cannula; *B*, contrast remaining in the saclike distended esophagus, and *C*, contrast in the ventricle (after one-sided vagotomy).

Thus, I include in groups 1 and 2 all cases with aspiration and its consequences, i. e., pneumonia, atelectases and pulmonary edema. (The consequences of anoxia for the cerebrum with edema, hemorrhage and fatty degeneration, I shall not discuss here.)

Group 3.—*Laryngeal Abductor Paralysis Combined with Two-Sided Pharyngeal Paresis.*—The central symptom in bulbar poliomyelitis is paresis of the pharynx, the mechanism of which I propose to examine

more in detail. The reason for this paresis is a nuclear lesion of the nucleus ambiguus, with resulting paralysis of the vagus.

One-sided pharyngeal paresis can be diagnosed by anamnesis. Many patients say they have noticed that it is easier to swallow food when lying on one side. Experience has shown that such patients often have a paresis on this side with a *signe de rideau* to the healthy side, a simple way of rapidly and early diagnosing a one-sided pharyngeal paresis. Sometimes no other symptom of bulbar poliomyelitis develops, and the patient is cured rapidly but keeps a *signe de rideau* for years (perhaps for life?).

In other cases, on the contrary, the one-sided pharyngeal paresis develops more or less quickly into a two-sided paresis with its well known symptom: absolute inability to swallow, with, at the same time, the gurgling sound in speech and respiration caused by retention of saliva in the pharynx.

This inability to swallow is due to paresis of the pharyngeal constrictors but also to a strong sympatheticotonic contracture of the cricopharyngeus muscle, and even the lower esophagus-ostium-cardia is often closed, as shall be noted presently.

If one does a one-sided vagotomy on an experimental animal (cat) high up in the neck under the tympanic bulla, a one-sided pharyngeal paresis results with a *signe de rideau* to the other side. If the vagus is also severed on the other side, there results a bilateral pharyngeal paresis with complete inability to swallow and, of course, a two-sided paresis of the vocal cords, with the plicae vocales standing still in a paramedian position alongside the median line. Such an animal dies of suffocation if a prophylactic tracheotomy is not done previously.

Figures 1 and 2 are taken from my experimental work of 1939.[6] In figure 1 is seen a doubly vagotomized and tracheotomized cat with contracture of the upper ostium (*A*), visible when the animal is, so to speak, in postural drainage, head downward. In figure 2 is seen the contracture of the cardia when the head is held up. One more fact appears from figure 2: The esophagus is strongly dilated, saclike, atonic and flaccid.

After this short digression, I return to some of the patients of my associates and me, who present an analogous picture.

These patients have a bilateral pharyngeal paresis but also have a two-sided abductor paralysis with the vocal cords standing still in paramedian position (exactly like those of the experimental animals!). If a tracheotomy had not been done, they would have been suffocated

6. (*a*) Sjöberg, A. A.: Experimental and Clinical Studies of Paresis of the Pharynx, Larynx and Oesophagus, Acta Otolaryng., 1939, supp. 38, p. 1. (*b*) Sjöberg, A. A., and Strömwall, E.: Experimentelle und klinische Studien über Schlund- und Kehlkopflähmungen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:322, 1940.

because of an inspiratory sucking in of the plicae vocales against the median line with obstruction of the rima glottidis (and possibly aspiration of saliva).

Dr. Wegelius, the hospital roentgenologist, took a few particularly instructive pictures of these cases. Dr. Wegelius and I intend, in a subsequent publication, to present a more detailed analysis when we shall have had the opportunity to follow the cases and have more experience.

Fig. 3.—Contracture of the ostium of the upper part of the esophagus, with flaccid, wide, piriform pockets filled with contrast medium.

In figure 3 is seen a contracture of the ostium of the upper part of the esophagus and the flaccid, wide, piriform pockets filled with contrast medium.

Figure 4 shows a dilated atonic esophagus with contracture of the cardia, as was seen in the animal experiments.

After tracheotomy, all these patients of ours recovered. They were given penicillin and dihydrostreptomycin and were soon without fever.

Between the seventh and tenth week we were able to decannulate them. The proper time for decannulation varies, though, usually, this depends on the degree of damage done to the muscles participating in the coordination of the complicated act of deglutition.

In this connection, I should like to draw attention to a fact often observed earlier, that the respirator is of no use in cases of what one used to call "central" respiratory paresis.

Fig. 4.—A dilated atonic esophagus, with contracture of the cardia.

Knowledge of the abductor paralysis explains some of these cases. Such patients, of course, cannot use the respirator. On the contrary, the respirator rather helps to suffocate them, since it compresses by suction the rima glottidis still more and at the same time sucks saliva into the larynx and the trachea. Only when a tracheotomy has been performed may the respirator be of use and, *nota bene*, if there is at the same time paresis of the respiratory muscles. In these cases tracheotomy must unconditionally be considered as lifesaving (and bronchoscopy is not sufficient).

GROUP 4.—*The Mechanism of Suffocation in Spinobulbar Lesions Combined with Two-Sided Pharyngeal Paresis.*—To this group belong, at least preliminarily, most of the cases.

It is well known, as I have stated in earlier publications,[7] that the virus of poliomyelitis has a curious tendency to produce a selective, disseminated destruction of various ganglia cell groups within both the medulla oblongata and the medulla spinalis.

In perhaps commoner cases is noticed a typical ascending paresis, beginning in the legs and traveling upward, thus producing a spinobulbar type. In other cases is seen a descending paralysis, resulting in a bulbospinal type. At times, an encephalitic element can be noted in the picture.

In these spinobulbar mixed types there is a very typical picture of suffocation. It cannot be too emphatically stressed that if the lives of these patients are to be saved they must be kept under constant observation and controlled carefully by trained observers.

For simplification, let me consider an ascending case. When the virus process has reached the thoracic cord, the intercostal musculature is paralyzed, and at the level of the third and fourth cervical segments the phrenic nerve is attacked and the diaphragm is paralyzed. At this stage, respirator treatment is indicated and can be of good use. The patient is now said to present a picture of peripheral respiratory paresis. If the process is arrested without ascending farther, life can usually be saved, thanks to the respirator.

Also in cases of only peripheral respiratory paresis, secretory aspiration can be fatal, and the patient can literally "drown" in his own secretions. Frequently repeated bronchoscopies with suction can be lifesaving.

At times, one is for various reasons obliged to perform first a tracheotomy and subsequently lower bronchoscopies.

But there can also be a sudden turn for the worse, owing to the destruction by the virus of new cervical segments also affecting the medulla and the vagus nuclei. The patient gets a typical two-sided pharyngeal paresis. In most cases the patient is by now so exhausted that the progress of the paralysis can no longer be analyzed.

Just before pharyngeal paresis sets in, one can, however, observe that often the patient has difficulty in moving his head and, especially, cannot always lift it from the pillow, in spite of the fact that the sternocleidomastoid muscles are not paretic. This inability seems instead to be caused by a disease of the cervical segment C_1 and C_2, by which the anterior neck muscles (sternothyroid, sternohyoid, omohyoid) and,

7. Sjöberg, A.: Experimental and Clinical Pareses of the Pharynx and Larynx, Nord. Med. **12**:3130 (Nov.) 1941: On Central and Peripheral Lesions in the Ninth and Tenth Cerebral Nerves, ibid. **19**:1398, 1943.

Fig. 5.—A tracheotomized man, with oxygen being supplied through the cannula. Notice the extreme sharpness of the angle between the bottom of the mouth and the anterior outline of the neck.

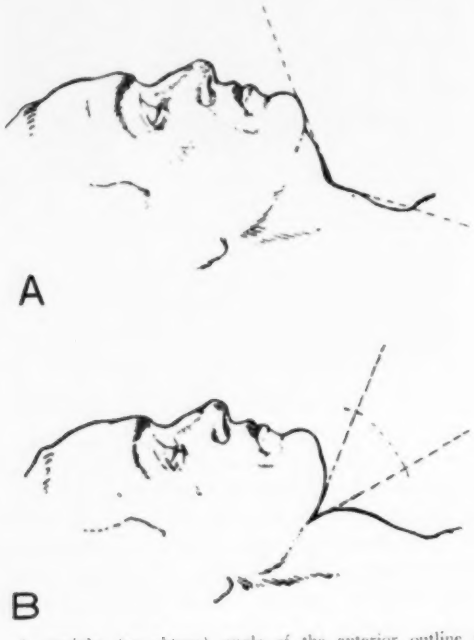

Fig. 6.—*A*, straight (or obtuse) angle of the anterior outline of the neck and the bottom of the mouth in a normal case. *B*, the angle symptom.

sometimes, the scaleni also are paralyzed. (It is known that the innervation passes over the cervical plexus and the ansa hypoglossi.)

In at least 7 of our cases I have been able to observe this mechanism. I will present here a concrete example that, at the same time, demonstrates a symptomatic picture, which, I think, has not been fully understood or described so far.

In figure 5 is seen a man lying tracheotomized, with oxygen being supplied through the cannula. Notice that the angle between the bottom of the mouth and the anterior outline of the neck is extremely sharp. Normally, this angle is straight or obtuse (fig. 6*A*).

In these pathologic cases, the angle is, on the contrary, extremely sharp (fig. 6*B*). In this connection I want to point out that this angle symptom is not influenced by the head being placed higher or lower.

Fig. 7.—Elimination of the angle symptom in convalescence.

Studies in normal cases show this fact plainly. It is also worth mentioning that the angle symptom is totally eliminated in the convalescent stage, when the pharyngeal paresis decreases and the mechanism of deglutition is again coordinated and proceeds normally. (See figure 7, which shows the patient in figure 5 convalescent, without the angle symptom.)

This angle symptom, or angle sign, seems to be caused by paralysis of the anterior neck muscles as well as of the pharyngeal constrictors. There results a contracture or tonus excess in the antagonists, i. e., in the remaining, nonparalyzed muscles, which normally maintain the larynx in position. Such muscles are, among others, the mylohyoideus (innervated by the fifth nerve), the digastricus (innervated by the seventh and fifth nerves) and the stylohyoideus (innervated by the seventh nerve). The result is that the larynx is pulled upward and backward, thus blocking the air passage to the trachea. Therefore, the

epiglottis does not fall back, but the opening of the larynx is pushed or pulled up under the base of the tongue, whereby the patient is suffocated.

The angle sign is still further accentuated if the accessorius nuclei are destroyed, in which case the trapezius and the sternocleidomastoid muscles on both sides are paralyzed, as is true of the patient shown in figure 8.

The young man in figure 8 has paresis of the trapezius as well as of the sternocleidomastoid. This is noted in the way he balances his head when he sits, because of that curiously extreme increase of nuchal

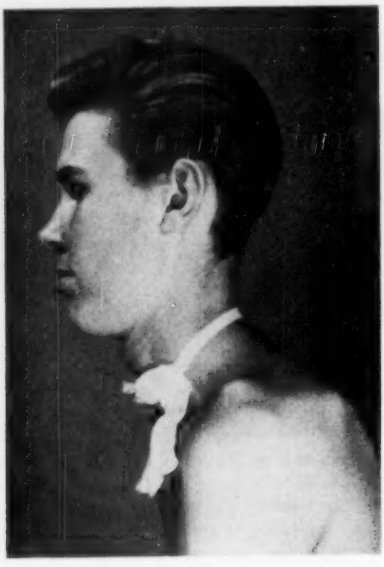

Fig. 8.—Paresis of the trapezius and the sternocleidomastoid muscle.

lordosis, caused by a contracture of the nonparalyzed antagonists (among others, the neck muscles which bend the head backward). The vertebrae of his neck assume an S-shaped, "swan neck" curve. On the roentgenogram (fig. 9) it is very clearly seen how the hyoid bone has been drawn upward and backward. The air passage is obstructed and the air shadow forms a sharp angle at the top (see *A*) in figure 9. The epiglottis touches the posterior wall of the pharynx.

In certain cases, one can see that the point of the angle is so sharp that a transverse skin fold, which divides the bottom of the chin from the neck, is extremely deepened. The neck appears as if cut off from

the bottom of the chin by a string (fig. 10). Judging from the outline, one would think that a thin running noose had been placed around the

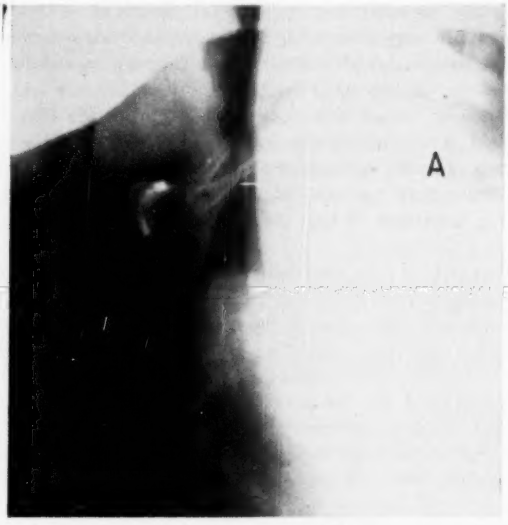

Fig. 9.—Roentgenogram showing how the hyoid bone has been drawn upward and backward, obstructing the air passage. *A* indicates sharp angle formed by the air shadow.

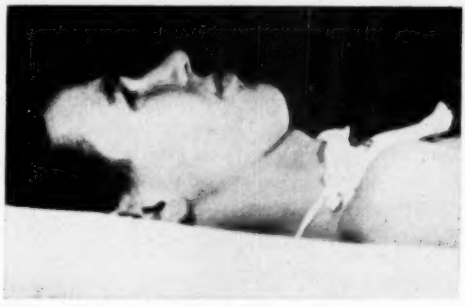

Fig. 10.—A sharp-pointed angle deepening extremely the transverse fold of skin, which is the dividing line between the bottom of the chin and the neck.

neck and pulled tight. In the depth of this fold one can palpate the hyoid bone. As can be seen from the roentgenogram, this indentation is probably due to the hyoid bone being dragged upward and, especially,

backward, owing to a contracture or tonus excess of the nonparalyzed antagonists (stylohyoideus and digastricus). The patients are literally strangled or hanged by those remaining muscles which are not paralyzed and which pull the hyoid bone upward and backward.

In men with a large, protruding thyroid cartilage this symptom is more marked than in women with a small, nonprotruding larynx skeleton. This angle symptom, or rope dent symptom, is therefore a warning signal from the patient himself that danger is imminent. He may suffocate any moment if a tracheotomy is not performed immediately or, if possible, before breathing becomes jerky and irregular. It is most useful for the nursing staff to know this symptom when observing a patient lying in the respirator, so that the laryngologist can be called immediately.

If such a patient is in the respirator the risk of suffocation is almost at its greatest, since the respirator has a tendency to obstruct still further the air passages in the region of the hyoid bone and the opening of the larynx, thus hastening suffocation. Breathing becomes shallow and jerky. The respiratory excursions become short and do not synchronize with the rhythm of the respirator. The face and lips become cyanotic. Cold sweat, peripheral coldness and rising blood pressure complete the picture. After tracheotomy these suffocation symptoms disappear rapidly and the patient feels relieved, getting quickly into the rhythm of the respirator.

The operation must be performed while the patient remains in the respirator. The operator must often work from behind, which is technically particularly difficult, and because of the often advanced spinal pareses, the trachea cannot always be satisfactorily lifted forward by a sufficient raising of the shoulders.

For certain reasons, I want to stress particularly that in many of our cases the operation was performed while the patient remained in the old Sahlin respirator with its lengthwise and crosswise rotating table.

The new ingenious Swedish Freiberger-Rudbeck [8] respirator hood has all the same great advantages. It is low, light and easy to manage and therefore more agreeable for the patient.

Now I operate mostly with the patient lying on the old Sahlin table but with the Freiberger hood connected with the Sahlin compressor aggregate.

I have been struck by the fact that in these cases, contrary to statements in the literature, there usually is no secretion or, in any case, only an insignificant amount of secretion in the bronchi, which observation must be considered to justify the importance attached to the angle, or rope dent, symptom in the diagnosis of a beginning suffocation.

8. Freiberger, Y., and Rudbeck, L.: A New Respirator, Svenska läk.-sällsk. förhandl. **48**:2623, 1949.

From this reasoning it also follows that etiologically and numerically the group of purely bulbar, purely "central" respiratory pareses is reduced in number. Instead, one has in most cases to do with spinobulbar mixed types. Characteristic of these types is a lesion localized in the first and second cervical segments, which suddenly combines with a nuclear lesion of the vagus, which in its turn causes a bilateral paresis of the pharynx. If, in addition to this, there is destruction of the accessorius nuclei, the situation becomes further aggravated.

I have, as a matter of fact, found cases which were at first considered as being purely bulbar and which, subsequently, after a tracheotomy, cleared and, permitting an analysis, proved to be "mixed" bulbar-cervical-spinal cases.

(Thus, we do not perform tracheotomies in all cases of paresis of the pharynx but only in those which—in the previously described way—are combined with cervical and spinal destruction.) These spinobulbar mixed types have, of course, the worst prognosis, but there is no doubt that the lives of these patients were saved through the operative treatment.

In the subsequent course of the illness there is the risk of continued hypoxia due to retention of secretions and, above all, to crust formation in the tracheobronchial tree and the blocking of, usually, the right main bronchus. The blood pressure must be taken constantly and seems to be a sensitive indicator of hypoxia. As soon as the blood pressure rises to 170 to 200 the patient becomes ashen gray, shows slight cyanosis of the face and the lips and is in a cold sweat.

Immediate bronchoscopy sometimes reveals hard obstructing crusts, which must be extracted with tongs or aspirated. (To combat crust formation we have started using instillations of 5 per cent sodium bicarbonate solution. At the same time we have noticed that carbogen [a mixture of 5 per cent carbon dioxide and 95 per cent of oxygen] does not dry up the mucous membrane so much as oxygen.) In other cases a purulent bronchitis appears with blocking of the bronchial branches. As soon as the bronchial toilet is concluded the blood pressure falls and the cyanosis disappears. The condition can hardly be attributed only to circulatory collapse but must primarily be due to hypoxia.

Once more I should like to stress the importance of postural drainage with head and trunk in a 20 to 30 degree incline downward (Galloway). Also, in the subsequent course of the illness, the table should, from time to time, alternately be rotated around the longitudinal axis to the left or to the right to allow the emptying of the different pulmonary segments.

"Bedside" roentgenograms, which can directly reveal an atelectasis or pneumonia, should be made. Thereafter, the obstructed bronchial branch

can be sucked clean through the bronchoscope, so that the pulmonary region becomes drained and pneumatized.

One of our cases illustrates the difficult nursing problem:

A man aged 21 with total paresis has been kept alive for more than two months and can now be considered to be out of acute danger. More than 30 bronchoscopies have been done, and in one night as many as five. Usually, one, two and three bronchoscopies were performed per day. He has also been under treatment with heparine sodium for a deep thrombosis in the right leg.

Bedside roentgenograms revealed, in addition, an atelectasis in the upper lobe of the right lung. After the upper lobe was cleansed by suction and micelle-naphazoline hydrochloride-sodium chloride solution instilled, it filled rapidly with air. He was decannulated after three months. The paresis of the pharynx was quickly compensated after three weeks.

The bacterial flora in the bronchial tree was carefully observed. Penicillin was given by injections and in aerosol in combination with streptomycin. The patient also received chloramphenicol (chloromycetin®).

Thanks to an energetic antibiotic therapy we have thus succeeded in bringing to a close our surgical treatment in this as well as in the other cases.

Group 5.—*Prophylactic Tracheotomy.*—The last question is: On what indications should tracheotomy be performed?

The answer for the future seems to be that tracheotomy should be performed early and prophylactically in serious cases. I think that this can best be explained by an example.

A 14 year old boy was seriously ill and showed "facies poliomyelitica" with hyperemia and slight cyanosis of the cheeks and circumoral triangular pallor. Examination on his arrival disclosed paresis of the arms, but no specific peripheral respiratory paresis was evident. He had, however, a certain air hunger, with jerky contractions of the auxiliary musculature, especially of the left sterno-cleidomastoid. There was one-sided paresis of the pharynx with beginning paresis of the other side but no paresis of the vocal cords. A tracheotomy was performed immediately on his arrival. In the night, total paresis of the pharynx set in and, also, paresis (peripheral) of the respiratory muscles, and he had to be put into the respirator where he soon "kept time." During the night his condition suddenly became almost shocklike. After he received a blood transfusion and postural drainage with suction and oxygen, his condition improved. After four weeks he could do without the respirator. The cannula was taken out after two and a half months and the paresis of the pharynx subsided after two months.

I consider this case an illustration of group 5, in which I also include early prophylactic tracheotomies. I am not yet ready to give a definite opinion in such cases. Our experience is much too limited, but I hope that the future will teach us more.

According to a procedure followed in the United States, we give liquid primarily in hypertonic and isotonic form; then dextran, plasma or blood transfusions are added. In other words, we have found that giving food through a nasal "feeding tube" should be avoided during

the first and, in certain cases, even during the second week because of the danger of aspiration. Later, when one has to resort to feeding by means of a probe, the probe should perhaps not reach lower down than the middle of the esophagus. It is best, before placing the probe, to determine with bedside roentgenograms and contrast medium in the esophagus whether the tonic cardiocontracture allows liquid to pass.

The problem which I have posed is complicated and difficult to master. It cannot be considered solved by performance of a technically difficult tracheotomy while the patient is under respirator treatment and by the introduction of a cannula into the trachea. Only then has the nursing problem been brought to its height, and the real difficulties begin to accumulate.

It must be remembered that the condition of these patients, whose lives have now been saved, was before considered quite hopeless, and they invariably died of suffocation. One deals here with paretic, profoundly unhappy, restless human beings, who are in imminent danger of atelectasis and pneumonia. Such a patient, and, as a matter of fact, all other tracheotomized patients, must have an intelligent, alert, specially trained nurse by his side night and day if his life is to be saved.

Since, so far, we lack another effective therapy which can forestall poliomyelitis or stop the virus destructions, we are obliged, because of the encouraging results hitherto attained, to continue on the newly opened path.

The seriousness and importance of the situation can be best demonstrated by quoting Seifert,[9] who made the following statement.

> I have seen and taken care of patients with poliomyelitis for eighteen years. Patients with bulbar poliomyelitis of the same type that we have seen in the last two and a half years have died under my care before this. It took a supervising nurse and a laryngologist to teach me that there was something to do about bulbar poliomyelitis.

Previously healthy people who are stricken with this ghastly type of infantile paralysis and who now, thanks to operative treatment, can be saved have in many cases a chance to recover a good working capacity and to be of use to society.

PRELIMINARY CONCLUSIONS

1. The majority of fatal cases in the early stages of infantile paralysis are, most probably, in accordance with the American opinion, primarily due to suffocation—anoxia—and in all probability not to circulatory paresis.

9. Seifert, M. H., in discussion on Galloway, T. C., and Seifert, M. H.: Bulbar Poliomyelitis: Favorable Results in Its Treatment as a Problem in Respiratory Obstruction, J. A. M. A. **141**:1 (Sept. 3) 1949.

2. According to Bergman and Huldt,[5] the mortality rate for patients treated with the respirator during the twelve year period (1934 through 1945) is 86 per cent (119 survived of 834) for the whole of Sweden and 82 per cent (28 of 158 survived) for Stockholm.

3. So far, thanks to the operative treatment, 12 of 14 patients whose condition has been considered hopeless up to now, have been saved.

4. The indications for operative treatment coincide with the immediate cause of suffocation and can be divided into the following five groups.

(*a*) Peripheral respiratory paresis with secretional retention.

(*b*) Aspiration (of saliva and vomitus) in purely bulbar cranial nerve lesions (above all, in paralysis of the vagus).

(*c*) Bilateral paralysis of the laryngeal abductors combined with two-sided paresis of the pharynx.

(*d*) Spinobulbar lesions, manifesting themselves, among other ways, in a bilateral paresis of the pharynx (plus, in certain cases, two-sided nuclear paresis of the accessory nerve, plus injuries to the first and second cervical segments of the spinal cord). The suffocation picture in these cases is dominated by the angle, or rope dent, symptom, which constitutes an alarm signal for the nursing staff. The patient runs the risk of being hanged or strangled any minute by a contracture or tonus excess in his nonparalyzed antagonistic muscles, which draw the hyoid bone backward and upward. If, in addition, a nuclear paresis of the accessory nerve sets in, the rope dent symptom is further accentuated and the patient's condition is aggravated, owing to a contracture of the nonparalyzed muscles which bend the head backward.

(*e*) Cases in which the operative treatment is applied early—prophylactically.

In all types of respiratory paresis, the respirator treatment may be directly harmful and can even hasten death by suffocation if bronchoscopic suction with or (in exceptional cases) without tracheotomy is not applied simultaneously.

Postural drainage with oxygen or carbogen is absolutely necessary. During the first, and sometimes even during the second, week the fluid balance must be carefully controlled and the liquid administered only parenterally in isotonic or hypertonic form. Dextran and blood transfusions have proved to have a beneficial effect on the initial shock condition, which often appears in the hours following a tracheotomy.

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TRACHEOTOMY IN POLIOMYELITIS

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DURING 1949 tracheotomies were done on patients with poliomyelitis in the New Haven Unit of the Grace-New Haven Community Hospital for the first time. The large number of tracheotomies was due both to a higher percentage of patients with severe paralysis requiring tracheotomy and to better understanding of the signs and symptoms of respiratory distress in these patients. The recent excellent articles by Galloway,[1] Priest [2] and others were instrumental in emphasizing the advantages of tracheotomy and the dangers of unrecognized anoxia. Our experience confirms their observations. Delay or failure to do a tracheotomy may result in needless fatality. We wish to record several pertinent observations.

The otolaryngologist is constantly seeing patients with various respiratory problems—croup, laryngeal cancer, foreign bodies in the air passages and postoperative atelectases. Consequently, he is well able to recognize signs of impending or existing respiratory distress. It is highly desirable, therefore, that the otolaryngologist be consulted early concerning every patient with poliomyelitis who has respiratory paralysis or signs of bulbar involvement.

Indications for tracheotomy are not always obvious. A patient with an apparently mild paralysis may have severe respiratory difficulty in an alarmingly short time, while a patient with bulbar signs that seem to be rapidly progressing may recover almost completely overnight. These patients require careful observation by a well trained team. At times there may be understandable reluctance on the part of the attend-

From the Department of Surgery, Division of Otolaryngology, Yale University School of Medicine.

1. (*a*) Galloway, T. C.: Tracheotomy in Bulbar Poliomyelitis, J. A. M. A. **123**:1096 (Dec. 25) 1943; (*b*) The Danger of Unrecognized Anoxia in Laryngology, Ann. Otol., Rhin. & Laryng. **55**:508 (Sept.) 1946. (*c*) Galloway, T. C., and Seifert, M. H.: Bulbar Poliomyelitis, J. A. M. A. **141**:1 (Sept. 3) 1949. (*d*) Galloway, T. C.: The Management of Respiratory Complications of Poliomyelitis, Arch. Otolaryng. **46**:125 (Aug.) 1947.

2. Priest, R. E.; Boies, L. R., and Goltz, N. F.: Tracheotomy in Bulbar Poliomyelitis, Ann. Otol., Rhin. & Laryng. **56**:250 (June) 1947.

ing physicians to approve performance of a so-called prophylactic tracheotomy on a patient whose symptoms may rapidly improve. At other times the patient may be so desperately ill that tracheotomy seems too dangerous a procedure.

A number of our patients were admitted with signs of spinal involvement and with sufficient respiratory paralysis to require a respirator. The disease then progressed and bulbar symptoms developed. In one fatal case the patient was in such poor condition that it was felt that removing him from the respirator to do the tracheotomy would be too great a hazard; so the operation was not done. In 4 other patients in poor condition the respirator was opened and artificial respiration was given by the anesthetists while the tracheotomy was performed. To an inexperienced team this may seem a precarious and difficult situation. In all 4 patients the actual performance of the tracheotomy under these conditions was surprisingly easy. Two of the patients died later, in

Summary of Data on Patients with Poliomyelitis Admitted to New Haven Unit of Grace-New Haven Community Hospital in 1949

	Total No.
Patients admitted for poliomyelitis	168
Patients with bulbar poliomyelitis	17
Tracheotomized patients	10
Deaths	6
Deaths in tracheotomized patients	5

spite of clear airways, from what was believed to be spread of the disease to the cardiac centers. The other 2 survived but were left with almost complete quadriplegia. After six months one of these patients is still unable to remain permanently out of the respirator.

In 6 of our cases the tracheotomy was done prior to putting the patient in the respirator. Since 5 of these patients complained of difficulty in swallowing their saliva, the tracheotomy would be more properly called therapeutic than prophylactic. Three of these patients died. In one of them, an adult, the tracheotomy was done on admission to the hospital. The patient's course went rapidly downhill, and he died six hours later in spite of a clear airway. The other 2 patients were children who complained on admission of much difficulty in swallowing their saliva. Since there was no respiratory paralysis and the lungs were clear, tracheotomy was deferred. One child had rapid progression of cyanosis and respiratory difficulty. Tracheotomy was done five hours after his admission but too late to affect the downhill course materially. The other child had cyanosis and flooding of the bronchi with saliva the second day. Tracheotomy was again done too late to save the child. Both children were put into the respirator

immediately after the tracheotomy and died a few hours later. Earlier tracheotomy might have saved these children. It is noteworthy that in all 3 children who died (the 2 mentioned previously and 1 other tracheotomized after being in the respirator) difficulty in swallowing was present on admission and cyanosis was allowed to develop before tracheotomy was performed.

PHYSIOLOGIC CONSIDERATIONS

The value of tracheotomy in combating cyanosis and flooding of the lower respiratory tract has been well shown by Galloway and others.[1] The cough mechanism can be considered to consist of five phases: (1) inspiration, (2) closure of the glottis, (3) increase in intrathoracic pressure, (4) opening of the glottis and (5) sudden expiration with release of the increased intrathoracic pressure. This mechanism may be interfered with in patients with poliomyelitis for the following reasons:

1. Motor paralysis of the larynx may prevent fixation of the vocal cords in the midline. This has been demonstrated by direct laryngoscopy in many cases [2] and interrupts the cough at phase 2.

2. Paralysis of the abdominal and intercostal muscles and the diaphragm will reduce the ability to increase intrathoracic pressure. To obtain an effective cough, the positive intrathoracic pressure may rise to 40 mm. of mercury.[3] Since the positive pressure in the respirator is usually set for a maximum of 5 mm. of mercury, this would not be sufficient in itself to produce an effective expiratory air blast. It is for this reason that prophylactic tracheotomy should be considered in respirator patients with severe paralysis of the chest and diaphragm even though no bulbar symptoms are present. The respirator patient who can cough effectively can usually close the glottis normally and does not have complete paralysis of the chest and diaphragm.

3. Hypoxia or encephalitis may cause central depression of respirations and of the cough reflex. Galloway [1a] has suggested that shallow respirations may be due to an effort by the patient to avoid aspirating pharyngeal secretions, since the respirations have been observed to deepen following tracheotomy.

4. Reduced sensitivity of the laryngeal mucosa has been suggested as another factor. We have not been impressed by any reduction in the sensitivity of the larynx and trachea. On the contrary, most of our patients have been acutely aware of mucus in the throat and have com-

3. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice: A University of Toronto Text in Applied Physiology, ed. 2, Baltimore, Williams & Wilkins Company, 1939, p. 484.

plained bitterly of it. Moreover, anesthesia elsewhere in the body is rare in poliomyelitis and one would not expect to find it frequently involving only the tenth nerve.

The swallowing mechanism may be interfered with for the following reasons:

1. There may be weakness or paralysis of the muscles concerned (mylohyoid, inferior constrictor of the pharynx, muscles of the tongue), and the patient may not be able to project the bolus into the esophagus at the usual speed.

2. Swallowing is a complicated mechanism requiring the coordination of several central nuclei. Disease in these centers may disturb such coordination.

3. The glottis is normally closed at the moment of swallowing. Paralysis of the cords may leave the glottis open and thus permit secretions and food to pass into the larynx.

4. The chest is fixed and air exchange is stopped during normal swallowing. The paralyzed patient cannot stop the air exchange while in the respirator. As a result, just as he swallows, the incoming air may carry liquids or food into the trachea. Most swallowing is done at a pause at the end of an expiration. There is no pause in the cycle of the respirator. Air is either coming in or going out. Perhaps it would be profitable to put an eccentric cam in the drive shaft of the respirator to give a short pause at the end of each expiration, which would permit swallowing at this phase of respirator cycle.

5. Swallowing while flat in bed is difficult at times for even a normal person.

6. Vomiting may be produced by interference in normal peristalsis of the esophagus or by irritation of the central nuclei.

INDICATIONS FOR TRACHEOTOMY

A.—Therapeutic tracheotomy should be considered whenever:

1. Any respirator patient exhibits signs of bulbar involvement or displays persistent cyanosis or struggling. (Restlessness and combativeness usually mean air hunger.) This includes all patients in the respirator with spinal involvement who have bulbar signs and all patients with bulbar poliomyelitis that progresses to respiratory involvement and who are put into the respirator.

2. Any nonrespirator patient is unable adequately to keep his airway free of secretions, as evidenced by moist rales, choking and cyanosis. Most nonrespirator patients with bulbar involvement will bitterly complain of their difficulty in swallowing and give plenty of warning of coming trouble.

B.—A prophylactic tracheotomy should be seriously considered whenever:

1. Early and progressive bulbar signs are present and before the patient has difficulty.

2. A long siege in the respirator seems probable, even though no bulbar signs are present. A patient with severe respiratory paralysis may have difficulty handling pharyngeal mucus because of an inability to cough effectively.

A certain number of these prophylactic tracheotomies might be life saving. However, it is impossible to forecast how many of the respirator patients will have enough difficulty to need a tracheotomy and some needless tracheotomies might be done. In such patients tracheotomy could be safely deferred until definite indications are present. Tracheotomy may be safely done at any time on any completely paralyzed patient as described later.

TECHNICAL CONSIDERATIONS OF TRACHEOTOMY

Since there usually is no laryngeal obstruction, it is not necessary to pass a bronchoscope before doing the tracheotomy. In an apprehensive, struggling patient such a procedure may even be undesirable. If the patient has advanced respiratory paralysis, respirations can be easily maintained by the use of a tight-fitting anesthesia mask, with oxygen insufflated by manual pressure on the bag in the system as illustrated (figure). It is not necessary to intubate the patient to use this technic unless the patient is unconscious. For even a completely paralyzed and desperately ill patient, the risks involved in actual tracheotomy are much less than the dangers of not doing a much needed procedure.

We have also used this technic of manual administration of oxygen to enable us to open the respirator and conduct a physical examination of the chest at leisure. Such examination is impossible with a noisy respirator operating or when the patient cannot inflate his own lungs. Frequent use of this simple technic has helped us to recognize early atelectasis when it was most effectively treated.

The time spent in changing the usual foam rubber collar of the respirator to the tracheotomy collar was often greater than the time needed for the tracheotomy. The special collar should be used whenever there is any possibility of a tracheotomy in the immediate future. All blood vessels should be carefully ligated, since the action of the collar rubbing on the neck may cause bleeding from a small vessel which would ordinarily not be of significance. Bleeding from a tracheotomy opening in a respirator patient can be (and was in one of our early patients) of great embarrassment. To aid in preventing mediastinal emphysema, it is better to avoid dissection of the fascial planes in the neck. We have

found it advisable to leave the tracheotomy tube open constantly at first. This not only by-passes the pharyngeal secretions, as shown by Galloway, which prevents their being drawn through the larynx, but it also enables the patient to swallow without the usual pause in the respiratory cycle. The latter factor may be of considerable importance in the relief afforded by the tracheotomy. We have also periodically plugged the tracheotomy tube and, by then using manual pressure on the thorax, helped the patient to cough more effectively. The nurses have been instructed to suction deeply into the bronchi occasionally and to suction

Technic of manual administration of oxygen.

deeply whenever there is mucus lower down that cannot be reached by ordinary suctioning. On the other hand, frequent deep suctioning may cause serious irritation and inflammation of the tracheobronchial mucosa. If the secretions are unusually thick and tenacious, instillation of 1 or 2 cc. of isotonic sodium chloride solution into the trachea before suctioning will often facilitate their removal. Since many patients are seen in the late fall and winter when the air indoors is dry, it may be helpful to install a humidifier in the room.

Removal of the tracheotomy tube should not be done until long after the patient is out of the respirator, since many of these patients have repeated episodes of atelectasis because of their poor ability to cough.

One of our patients showed atelectasis after she was extubated. Treatment of the atelectasis was much more difficult because of the absence of the tracheotomy tube. It would be better to err on the side of leaving the tube in too long than to risk such a situation. The tubes should not be plugged at first. Later on, when the acute stage of the disease has passed, the tube may be plugged for comfort or when the patient wishes to talk. During coughing the tube may be plugged or occluded with a finger by the nurse. This sometimes improves the effectiveness of the cough. It is also occasionally beneficial to help the patient to cough by using manual pressure on the thorax, especially after the patient has been removed from the respirator. Many are able to stay out of the respirator long before they have a really effective cough. The additional manual pressure enables the patient to build up a higher intrathoracic pressure and thereby cough more effectively.

These patients represent a most difficult problem in nursing care. Only competent nurses, well trained in the care of tracheotomized patients, should be assigned to them. An inexperienced nurse may permit an episode of cyanosis to remain untreated, and even one such short episode may lead to a fatal outcome.

ANALYSIS OF CASES

It has been shown that in poliomyelitis damage can and does occur to the respiratory and circulatory centers in the reticular substances.[4] The prognosis is much worse in these cases and some patients will die regardless of any measures taken to save them. In 2 of our patients who died, we felt that involvement of the circulatory centers had occurred. In 4 of them an early tracheotomy might have been life saving. Priest, Boies and Goltz reported a mortality of 60 per cent among 75 tracheotomized poliomyelitis patients. We have had a 50 per cent mortality among 10 such patients. In none of these patients was death in any way attributable to the operative procedure itself. Though we have not done the tracheotomies with a bronchoscope in place, we have not encountered pneumothorax or interstitial emphysema, probably because none of our patients had any laryngeal obstruction. Because all our tracheotomized patients were in the respirator and also had difficulty in swallowing, we felt that the tracheotomy was probably life saving for every one who survived. These patients all felt that the tracheotomy had been of great value to them, and several emphasized the terrible feeling of suffocation they had had when they were unable to clear their throats of the accumulated mucus.

4. Baker, A. B.; Matzke, H. A., and Brown, J. R.: Poliomyelitis, Arch. Neurol. & Psychiat. **63**:257 (Feb.) 1950.

SUMMARY

The otolaryngologist should be consulted immediately concerning every patient having poliomyelitis with symptoms of bulbar involvement or respiratory difficulty.

In a desperately ill patient in need of a tracheotomy, it is probably more dangerous to delay the tracheotomy than to perform it.

Various physiologic considerations of the cough and swallowing mechanisms are reviewed.

Tracheotomy was done on 10 patients with bulbar poliomyelitis. Five of these patients died.

In all surviving tracheotomized patients, the tracheotomy was probably a life-saving procedure. Earlier tracheotomy in 3 of the 5 who died might have been life saving.

REPORT OF CASES

CASE 1.—S. F., an 8 year old boy, was unable to swallow, vomited continually and had a thin, weak voice. Five hours after his admission to the hospital, respirations became shallow and rapid and retractions of the ribs were noted on inspiration. Many moist rhonchi were heard and the child showed cyanosis. Tracheotomy was done and much mucus was aspirated from the trachea, with rapid clearing of the chest. Cyanosis again developed in three more hours, and the child was put into a respirator but died almost immediately.

Comment.—Earlier tracheotomy was indicated.

CASE 2.—J. G., a 12 year old boy, had rapidly progressing bulbar poliomyelitis. He was unable to swallow and complained of much trouble with pharyngeal mucus on his admission to the hospital. Tracheotomy was deferred. Respiratory paralysis and cyanosis began to develop on the third hospital day. His color remained poor even after he was put into a respirator. A tracheotomy was done with some temporary improvement, but he died shortly thereafter.

Comment.—Earlier tracheotomy was needed.

CASE 3.—M. D., a 6 year old girl, had rapidly progressing bulbar poliomyelitis. She was unable to swallow and complained of much trouble with pharyngeal mucus. Tracheotomy was deferred. Respiratory paralysis began to develop twenty-four hours after her admission, with moist rales, cyanosis and shallow respirations. Tracheotomy was done and much mucus was aspirated from the trachea. Her condition improved a little when she was placed into the respirator, but she died four hours later.

Comment.—Earlier tracheotomy was needed.

CASE 4.—P. M., an obese 19 year old woman, was admitted to the hospital with severe spinal involvement, severe respiratory paralysis, a slurred, weak voice and a weak cough. Immediate prophylactic tracheotomy was done and she was put into a respirator. She did well for twenty-four hours but died suddenly in spite of a clear airway and no cyanosis.

Comment.—This patient probably had cardiac arrest.

CASE 5.—E. E., a 36 year old woman, was admitted to the hospital with mild spinal paralysis and mild respiratory difficulty three weeks post partum. (Her 5 year old daughter had had mild bulbar poliomyelitis one week previously.) She

had increased respiratory distress and difficulty in swallowing on the fourth day in the hospital. Tracheotomy was immediately performed and she was put into a respirator. Her condition gradually improved and she began eating on the seventh day. She was out of the respirator on the twentieth day, after having had several short episodes of atelectasis. She was extubated on the twenty-fourth day. She complained of persistent inability to cough effectively and on the thirty-third day had another episode of atelectasis which cleared in several days.

Comment.—Tracheotomy was of much value. The tube was removed too soon.

CASE 6.—C. B., a 40 year old man, had severe spinal paralysis on his admission to the hospital. On the third hospital day he displayed increasing respiratory distress and difficulty in swallowing mucus. Tracheotomy was done immediately and he was put into a respirator. He began to swallow better on the ninth day and was out of the respirator on the sixteenth day. He had several subsequent short episodes of atelectasis. The tube was removed in the ninth week.

Comment.—Tracheotomy was of much value.

CASE 7.—E. P., a 42 year old woman, was admitted to the hospital with moderate spinal paralysis, fuzzy speech and mild difficulty in swallowing. (An 8 year old daughter died in another hospital of bulbar poliomyelitis after one day's illness.) The patient had no respiratory difficulty and her lungs remained clear. On the third day she complained of increasing difficulty in swallowing mucus. A tracheotomy was done, with considerable relief. The next day respiratory paralysis swiftly developed and she became comatose. She was put into the respirator, with rapid clearing of her sensorium. She required intravenous administration of fluids for thirty days and was fed through a stomach tube for another month. She has had persistent midline paralysis of her vocal cords to the present and is unable to breathe without the tracheotomy tube.

Comment.—Tracheotomy was of great value.

CASE 8.—E. P., a 40 year old woman, was admitted to the hospital with moderate spinal paralysis and mild paralysis of the diaphragm and intercostal muscles. She was put into a respirator immediately and did well at first. She began to complain of difficulty in swallowing on the twelfth day. On the fourteenth day she had mild cyanosis and roentgenographic evidence of atelectasis and pneumonitis of the right lung. Bronchoscopy was done with the patient in the respirator, and some mucus was aspirated from the right bronchus. A tracheotomy was then done and was followed by rapid improvement. She had several subsequent episodes of atelectasis which cleared with suctioning, postural drainage and chemotherapy. She was out of the respirator after four months and was extubated after six months.

Comment.—Tracheotomy was of great value.

CASE 9.—A. H., a 41 year old man, was admitted to the hospital with moderate spinal paralysis and moderate paralysis of the chest and diaphragm. He was put into the respirator immediately. There was complete peripheral paralysis by the second day, and he began to complain of difficulty with mucus. Even though he could still swallow fluids, tracheotomy was done on the third day because of increasing difficulty with pharyngeal mucus. He did well following this but had several episodes of atelectasis during the next four months. After six months, he is still unable to remain out of the respirator completely.

Comment.—Tracheotomy was of great value.

CASE 10.—C. O., a 19 year old woman, was admitted to the hospital with severe spinal and respiratory paralysis. Her voice was thin but she could swallow without difficulty. She was immediately put into a respirator. On the second day she was

completely unable to swallow, so a tracheotomy was done. Mucus was present in the trachea. On the third day she had severe abdominal distention and frequent vomiting, as well as increased peripheral paralysis. On the fourth day her condition showed a rapid downhill course in spite of frequent suctioning and a clear airway.

Comment.—Tracheotomy failed to prevent death. The patient probably had cardiac arrest.

CASE 11.—E. W., a 31 year old man, was admitted to the hospital with mild spinal paralysis, no bulbar symptoms and no respiratory paralysis. On the second day he was put into a respirator because of increasing respiratory paralysis. On the fourth day he had complete peripheral paralysis, complained of difficulty in swallowing mucus and had mild cyanosis. Because of his critical condition, tracheotomy was deferred. He was treated with frequent suctioning and postural drainage. His course was progressively downhill and he died on the eighth day. Postmortem examination revealed pulmonary edema and acute meningoencephalomyelitis.

Comment.—Tracheotomy might have saved this patient.

ARTERIOVENOUS ANEURYSM OF THE INTERNAL CAROTID ARTERY IN THE CAVERNOUS SINUS

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ARTERIOVENOUS aneurysm of the internal carotid artery in the cavernous sinus is not a rare condition—not even an uncommon one—since the literature already shows about a thousand authentic cases. This case is being reported because it was discovered inadvertently when the patient came for treatment of a polypoid ethmoiditis. It is of interest to otolaryngologists not only for that reason but also because it is one more cause for obscure nasal bleeding, which should be borne in mind when refractory nasal hemorrhage occurs with source and cause not easily recognized.

On Oct. 6, 1942, I saw for the first time a married woman aged 48 who desired more permanent relief from a bilateral polypoid ethmoiditis, for which she had had frequent removals of polypoid tissue for temporary comfort. Incidentally, she mentioned that for six or eight weeks she had been conscious of a continuous pumping sensation anterior to her right auricle, which was quite bothersome at night. She had never had any pain in relation to her ears but was aware that her hearing was not entirely normal. It is not uncommon to have pulsation in relation to the ear, especially when there are large arteries in the vicinity of the eighth nerve and pulsation transmitted through the ear itself, so that not too much stress was laid on this symptom at this time. Naturally, all interest was focused on the ethmoidal condition, relief from which she desired. On examination, both drum membranes were retracted and her hearing was characteristic of a chronic catarrhal otitis media. Nasal examination revealed a typical bilateral polypoid ethmoiditis, with quite complete obstruction. Roentgenograms of the sinuses were diagnosed as "cloudy antra and ethmoids, especially most marked in the right posterior ethmoid region."

A bilateral ethmoidectomy was advised and arranged for but delayed for two months, since there was no great urgency and because the patient's busy season as a milliner would be over by the first of December. This delay was particularly fortuitous for the patient and myself, because

Read at a meeting of the New England Otolaryngological Society, February 1950.

during this waiting period other symptoms which were of much greater importance supervened. Three days before the date for operation the patient came to see me because for ten days her right eye had been protruding and she had been conscious of a swishing sound behind that eye. She had no headache or nerve paresis but did have a typical pulsating right exophthalmos with chemosis. The motions of the eye were perfectly free, although there was some proptosis. It became evident that this situation probably represented a vascular condition, and, with the stethoscope placed over her right antrum, it was possible to hear a bruit and the swishing sound of which she had complained. There was no doubt that this was an arteriovenous aneurysm, but its exact location was not easily recognized. An ophthalmologic examination confirmed this opinion and, further, supplied the information that the optic nerve and the third, fourth and sixth nerves were unaffected. The patient was referred to the hospital, where she was given a complete examination. The results of skull plates, neurologic and spinal examination and examination of the blood—both for cell counts and serum tests—were all within normal limits. The patient did have a 180 systolic and 100 diastolic blood pressure. Further history gave no evidence of trauma as a background, but family history later elicited the fact that her father had died of an aortic aneurysm.

It was felt at the time that eventually the common carotid artery would have to be ligated, but the dangers of ligation at her age and with her hypertensive condition would have to be borne in mind. Because there was no paresis it was uncertain whether this arteriovenous aneurysm was in the cavernous sinus or, rather, more posteriorly, in the ophthalmic artery. It was considered, since it had probably broken into a vein, that the aneurysm was fistulous, that it might even thrombose and would not become large. The posterior nasal polypoid obstruction seemed to be very large, in the opinion of the neurologist, neurosurgeon and ophthalmologist. The medical consultant likewise was inclined to feel that removal of the obstruction should be the first move. Another, independent, neurosurgical consultation confirmed this opinion. A waiting period was decided on, during which a course of digital compression of the common carotid artery in the neck was started. The patient was allowed to go home but was advised to remain under close observation.

On December 8, about five days later, she reported that the swishing sound was gone, that the eye was definitely receding and that the pulsation was less loud. At this time there was about 5 mm. of exophthalmos. Ten days later she was still improving and the consensus was that the aneurysm would not rupture any further, for which reason there was more of a tendency to delay ligature of the carotid artery, for fear lest anemia of the brain and consequent hemiplegia, with attendant circumstances, might be brought on.

On Feb. 1, 1943, with general anesthesia, a bilateral ethmoidectomy was carried out uneventfully. There was a fair amount of oozing postoperatively for eight hours, but the relief in nasal breathing was manifest and a source of much comfort to the patient. However, four days later, the exophthalmos began to increase very definitely. This was thought at first to be due to reaction attendant on operation or, possibly, increased thrombosis in the orbital vessels. This belief seemed to be justified, because in a few days the exophthalmos again began to recede, and on February 8 the patient was able to leave the hospital. On February 16—

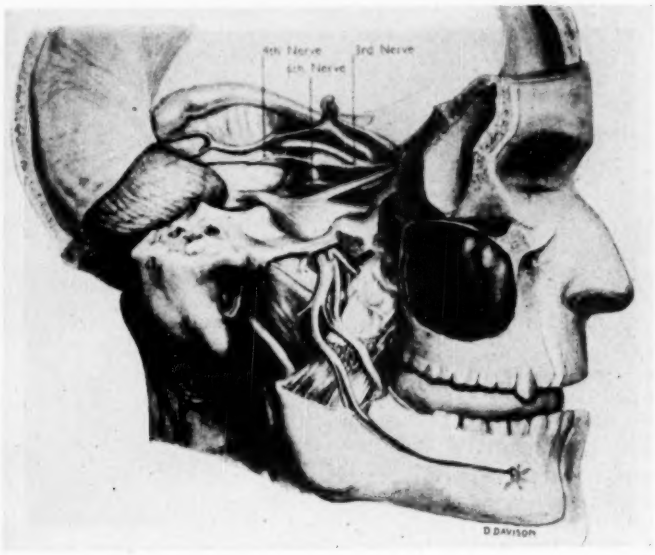

Fig. 1.—Lateral view of cavernous sinus with carotid artery and third, fourth, fifth and sixth nerve relations. (From Jefferson.[2])

sixteen days after the ethmoidectomy—she returned with much more definite exophthalmos. The protrusion now measured between 11 and 12 mm., and there were some retinal hemorrhages. The ophthalmologist now felt that the time was ripe for ligature of the common carotid artery. The patient had been practicing digital compression all this time. By this time all concerned were agreed that ligature should now be carried out.

On March 4 the patient entered the hospital. A percutaneous injection of colloidal thorium dioxide (thorotrast*) was made into the carotid

artery according to the Loman-Myerson[1] technic, and roentgenograms were taken, which definitely showed the arteriovenous aneurysm, presumably in the cavernous sinus. The injection showed the course of the internal carotid artery and where the fistula had occurred, with mingling of the arterial and venous blood, definitely proving that it could be only a small fistula not causing any great distention or pressure on the third,

On March 6, with local anesthesia produced by procaine hydrochloride (novocain®), the common carotid artery was isolated in the lower half of the neck; an aluminum band was placed about the artery, and it was compressed sufficiently to cause about 80 per cent of occlusion

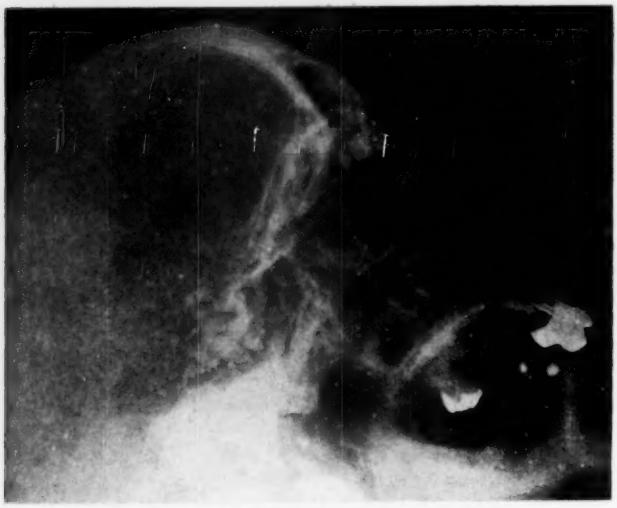

Fig. 2.—Visualization of the fistulous arteriovenous aneurysm in the cavernous sinus, with mingling of the arterial and venous blood.

in the lumen of the vessel. After the patient was observed for about fifteen minutes without any untoward symptoms or evidence of hemiplegia being noted, a silk thread was placed below the aluminum band, in order more easily to identify the vessel later, and the wound was closed with a short strand of the silk thread protruding outside the closure. The patient was returned to bed in good condition.

1. Loman, J., and Myerson, A.: Visualization of the Cerebral Vessels by Direct Intracarotid Injection of Thorium Dioxide (Thorotrast), Am. J. Roentgenol. (Feb.) 1936. Freedman, L. M.: Puncture of the Internal Jugular Vein in Cases of Mastoiditis, Arch. Otolaryng. **23**:29 (Jan.) 1936.

For forty-eight hours she did very well; the eye began to recede almost immediately. However, on the third postoperative morning, at 10 o'clock, without any warning, she suddenly became apathetic; the vision became somewhat cloudy, the face showed muscular weakness in the lower half of the left side, and there was weakness of the left arm—all evidence of anemia of the brain. It was assumed that postoperative edema at the site of operation had converted the partial occlusion to a complete one. It was thought best to observe the patient before doing anything more active. This judgment proved to be quite salutary, because by the afternoon the paresis began to recede. Homonymous hemianopsia had developed, which cleared up quickly, and by the next morning all the untoward reaction had disappeared. The face had returned to normal, the weakness of the arm likewise had disappeared and vision was normal; it had been only a fleeting condition. On March 22, the eye having receded to its normal position in the orbit, the wound was reopened with local anesthesia, the vessel tied below the clamp and the clamp removed. The vessel was tied above the position of the clamp but was so friable that the suture cut through and some terrifying bleeding occurred before the upper segment of the vessel was found and secured by another ligature. She had an uninterrupted course to recovery, and she left the hospital one week later. She was observed until June of 1948, and when last seen was in perfect condition. Recently she reported herself free from symptoms.

A review of the literature with relation to intracranial carotid aneurysm is very interesting. Since this condition is not very commonly seen, it apparently is quite reasonable to assume that it is a rare condition. This assumption is far from true. In fact, the condition is relatively common. Jefferson,[2] in 1938 and 1939, reported a series of cases and called attention to the fact that aneurysm of the internal carotid artery is rare in its cervical portion but common in its intracranial portion. Smaller aneurysms are prone to rupture and cause subarachnoid hemorrhage, which may be widespread without giving signs as to their exact site; large ones behave more like tumors, especially after the hemorrhage has ceased and thrombosis occurs. This is more characteristic of the types that sacculate and do not completely break through as fistulas. Some may remain stationary for years, but, by pressure on nerves largely in relation to the branches of the trigeminal nerve and the third, fourth and sixth nerves, they may cause permanent paralysis. Jefferson had collected a series of 55 cases.

He predicated his conclusions on the fact that the internal carotid artery, once it enters the cranial cavity, no longer has as much fascial and adventitious support as it has in its extracranial portions and

2. Jefferson, G.: On Saccular Aneurysms of Internal Carotid Artery in Cavernous Sinus, Brit. J. Surg. **26**:267 (Oct.) 1938.

thus is most easily susceptible to aneurysmal weakness of its wall. The three commonest causes, according to his analysis, are syphilis which is today comparatively uncommon; trauma, which can always be a prominent cause, and, finally, congenital origin, which is quite frequent. In the congenital types, family history is most important.

He classified four common types of internal carotid aneurysm in relation to the cavernous sinus.

1. The apoplectic type, which gives rise to subarachnoid hemorrhage and no other signs.

2. The fistulous type, with isolated paralyses according to involvement of the third, fourth and sixth nerves and branches of the fifth.

3. The saccular type, nonfistulous in the cavernous sinus, with nerve paralyses according to position.

4. Basal aneurysm, which affects the visual pathways and is external to the cavernous sinus.

The saccular, nonfistulous type develops in the anterior, middle or posterior portions of the cavernous sinus, and each can be distinguished by the nerves involved. The anterior saccular aneurysm would affect only the first branch of the trigeminus; the middle would affect the first and second branches of the trigeminus, and the posterior would affect all the branches of that nerve. It all depends on how large the sacculation is and how much it distends into the cavernous sinus, compressing the nerve branches. The paralyses of the third, fourth and sixth nerves depend entirely on the degree of compression from distention of such sacculation.

It is not within the scope of this paper to go much further into these distinctions. What is of especial interest for us is the realization of how it may concern us as otolaryngologists. Jefferson described amongst the cases he listed a case with paroxysmal pain and nasal bleeding which was found at operation to be caused by such an aneurysm. He described another case with anesthesia of the face, defective vision and enlargement of the sphenoid fissure, which might have broken into the nose. Another case of especial interest was that of a 34 year old man operated on for polypoid ethmoiditis, who, three weeks later had a violent hemorrhage from the nose, losing a pint of blood in two or three minutes. He had four such hemorrhages in the following week, required a number of transfusions and, during the last bleeding, was unable to hold any packs in his nose because of the violence of the hemorrhages. Finally, the left carotid artery was tied off, after which he had facial paralysis on the right and aphasia; these improved but never entirely disappeared. They were due to an aneurysm which broke through into the sphenoid sinus, illustrating the penalties of carotid ligation.

Locke,[3] of San Francisco, found 588 cases in the literature from 1809 to 1907. He analyzed the cases in much the same manner as did Jefferson in his smaller series. In his series of 588 cases he found 23.16 per cent of the spontaneous type and 76 per cent of traumatic origin. The spontaneous type predominated in women, and the average age at which this type occurred was more in the fifth decade and after. He found arteriosclerosis prominent in the spontaneous cases; only a few cases of syphilis were present. Practically all the traumatic cases were arteriovenous, of the internal carotid or ophthalmic arteries. He, like Jefferson, explained the nerve paralysis as depending on the size of the aneurysm, fistulization or sacculation. In the congenital type he found

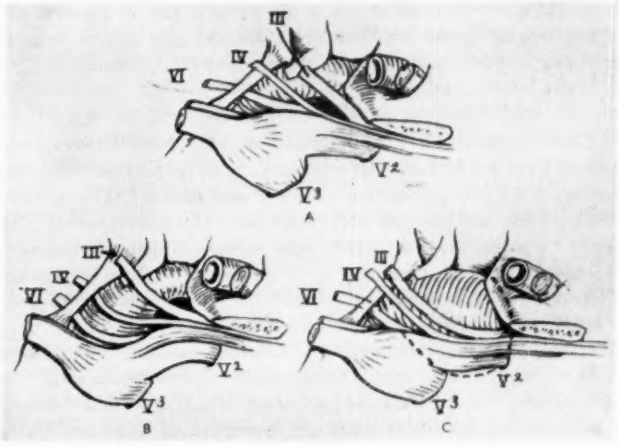

Fig. 3.—Diagram of the anatomic syndromes of the cavernous sinus; *a* indicates a normal sinus; *b*, a syndrome of the posterior end of the cavernous sinus, and *c*, a middle syndrome of the cavernous sinus. (From Jefferson.[2])

50 per cent of the cases to be arteriovenous, 25 per cent of tumor, 25 per cent of aneurysm alone— of the internal carotid or the ophthalmic artery. He explained the mechanism of the pulsating exophthalmos as due to weakened arterial walls in the spontaneous cases. There were also cases of bilateral exophthalmos due to spread through the circular sinuses, anteriorly and posteriorly. In relation to the treatment, he advised compression and/or ligation of the common carotid artery. It was important to decrease the blood going to that side of the brain without diminishing the blood supply to the brain, in order to prevent hemorrhage or death.

3. Locke, C. E., Jr.: Intracranial Arterio-Venous Aneurism or Pulsating Exophthalmos, Ann. Surg. **80**:1; 272, 1924.

Prolonged digital compression in some cases stopped the bruit. In the young, he considered it unwise to carry on digital compression too long lest subsequent carotid ligation might increase the anastomosing circulation too much. If digital compression shuts off the bruit and causes headache or sensory signs on the opposite side, a thorough slow course of digital compression is indicated. It is especially important in the elderly or middle-aged. If carotid compression does not stop the bruit or cause sensory or other signs of brain anemia, then compression—however prolonged—will be useless. He advised a three week course of compression before ligature. There is little choice between internal carotid and common carotid ligation as to end result, except that the mortality rate is higher in cases of internal carotid ligation. The common carotid artery is assumed now to be the safer site for ligature, since it is less frequently the site of sclerotic plaques and because it allows some retrograde circulation through the external carotid artery.

SUMMARY

A case report is here presented of a spontaneous arteriovenous aneurysm from the internal carotid artery, in the right cavernous sinus, occurring in a woman of early middle age who gave a family history of aneurysm. She had marked arteriosclerosis. The first symptom was a sense of pulsation anterior to the right auricle, which was mentioned only incidentally and had been going on for several weeks when she came for consideration of her polypoid ethmoiditis. For reasons unrelated to her condition, the ethmoidectomy was delayed for two months, during which time pulsating exophthalmos, bruit and swishing sounds supervened. It was possible, then, to make the diagnosis which might have been obscured by the ethmoid operation and might have provoked the question whether the cause of the arteriovenous aneurysm was not in some way related to the operation. Eventually, the bilateral ethmoidectomy was carried out successfully and without serious incident, which was very fortunate. A few weeks later, when the exophthalmos became more prominent, and with retinal hemorrhages supervening, after a sufficient period of practice with digital compression, ligation of the common carotid artery was carried out. There was a fleeting reaction, with slight facial paralysis and partial hemiplegia, lasting about six or eight hours. From then on the eye receded to its normal position and all symptoms disappeared within one week. The patient has remained without symptoms to this date.

EFFECT OF AUTONOMIC DENERVATION ON NASAL MUCOSA

Interruption of Sympathetic and Parasympathetic Fibers

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IN ADDITION to the nerves of ordinary sensation, the mucous membranes of the nose and paranasal sinuses are innervated by both sympathetic and parasympathetic fibers. In the past several years, the department of neurosurgery at the Cleveland Clinic Hospital has been carrying out operative procedures which in some cases interrupt the sympathetic nerve supply to the head and in other cases the parasympathetic nerve supply.[1] These procedures have afforded us an opportunity of observing the effect on the nasal mucosa of sympathetic denervation on the one hand and parasympathetic denervation on the other.

The parasympathetic fibers leave the brain stem as the nervus intermedius, which constitutes a portion of the facial nerve. At the geniculate ganglion some of the parasympathetic fibers leave the facial nerve to form the greater superficial petrosal nerve, which passes to the sphenopalatine ganglion; from there, postganglionic fibers go with the deep branches of the maxillary division of the trigeminal nerve to the mucous membrane of the nose and paranasal sinuses.

The sympathetic outflow to the head has its cells of origin in the intermediolateral column of the upper thoracic segments of the spinal cord. The preganglionic fibers pass by way of the anterior thoracic roots through the stellate ganglion mainly to the superior cervical ganglion. From here postganglionic fibers pass by way of the carotid plexus and deep petrosal nerve to the sphenopalatine ganglion and from there to the mucous membrane of the nose and paranasal sinuses, also

From the Cleveland Clinic and the Frank E. Bunts Educational Institute.

1. Gardner, W. J.; Stowell, A., and Dutlinger, R.: Resection of Greater Superficial Petrosal Nerve in Treatment of Unilateral Headache, J. Neurosurg. **4**:105 (March) 1947. Gardner, W. J., and Williams, G. H., Jr.: Interruption of Sympathetic Nerve Supply to Brain—Effect on Parkinson's Syndrome, Arch. Neurol. & Psychiat. **61**:413 (April) 1949. Karnosh, L. J., and Gardner, W. J.: Effects of Bilateral Stellate Ganglion Block Depression, Cleveland Clin. Quart. **14**:133 (July) 1947.

with the deep branches of the maxillary division of the trigeminal nerve. The parasympathetic fibers are generally vasodilator and secretomotor in function and are concerned primarily with the functional regulation of the mucous and serous glands in the nasal mucous membrane. The vasoconstrictor reactions in the mucous membranes are regulated through the sympathetic innervation.

A series of 27 cases was available for study in which the sympathetic supply to the head was interrupted surgically in the course of treatment of a variety of neurologic disorders. Each case was studied with particular attention given to the presenting nasal symptoms and the physical changes seen in the mucous membrane of the nose. The relative frequency of infections of the upper respiratory tract in each case was compared both preoperatively and postoperatively in an attempt to determine any change in individual susceptibility. The cervical sympathetic chain was interrupted by one of three procedures. Bilateral superior cervical ganglionectomy was performed in 8 cases. Bilateral stellate ganglionectomy was performed in 10 cases, and bilateral anterior thoracic rhizotomy with section of the accompanying sympathetic efferents to the first through the sixth thoracic nerves was performed in 9 cases. Observation was made in the majority of these cases during a period of six months to two years after the surgical procedure. Progress of these patients after this time was not observed except in a few cases.

In addition to those in this series, 6 patients were observed in whom the parasympathetic supply to the nasal cavity was interrupted surgically. This surgical procedure consisted of section of the greater superficial petrosal nerve just beyond the hiatus Falopii on the floor of the middle fossa of the skull. The operation was performed for the relief of unilateral headache. In this group the surgical procedure was unilateral and it was possible to compare the normal side with the parasympathectomized side. Attention was again given to the presenting nasal symptoms and the physical changes in the nasal mucous membrane.

Bilateral superior cervical ganglionectomy, as was stated previously, was performed in 8 cases. This procedure constitutes a postganglionic sympathectomy. All patients in this group presented symptoms of excessive nasal stuffiness with increased nasal discharge. The symptoms of nasal stuffiness varied in degree in different patients as well as in the same patient.

In most cases the nasal stuffiness was constantly present to some degree but varied in severity from time to time. Several patients complained particularly of symptoms when they were in the recumbent position. The amount of nasal discharge, although increased, was not of copious quantity, and most patients minimized this symptom. The

quality of the discharge varied in different patients from a thin serous to a mucoid type of drainage. None of the patients in this group had had any nasal symptoms or complaints prior to removal of the superior cervical ganglion. These symptoms, which began immediately after the surgical procedure, were still present in about the same degree after eighteen months to two years. There was no apparent change in the individual susceptibility to infections of the upper respiratory tract after the surgical procedure. The patients in this group had on an average of one such infection a year prior to operation and noticed no increase in frequency of infections following operation. The examination of the nasal cavity revealed similar findings in all cases except for minor structural variations. The mucous membrane covering the septum and the turbinate bodies was swollen and somewhat more pale than normal. An excessive amount of nasal secretions was present, varying from a serous type to a thicker mucoid variety. In no instance was purulent exudate found. There was no evidence of polypoid changes in the mucous membranes, and no true polyps were found. There was no apparent change in the appreciation of pain and pressure over the various parts of the mucous membrane. The sense of smell remained normal. The response of the mucous membranes to a locally applied vasoconstrictor (0.25 per cent phenylephrine hydrochloride solution [neosynephrine hydrochloride*]) was prompt and complete and afforded temporary relief from the nasal symptoms. A biopsy of the mucous membrane covering the middle turbinate body was obtained from one of this group. The microscopic section showed evidence of chronic inflammation of mucous membrane. The glandular elements were normal in appearance with a tendency toward somewhat hyperplastic mucous glands. The surface epithelium was of the normal pseudostratified ciliated columnar type.

The second group of patients studied were those in which the bilateral stellate ganglionectomy was performed. This procedure constitutes a preganglionic sympathectomy of the nasal mucosa. A total of 10 patients were seen in this group and again all had symptoms of nasal stuffiness of variable degree. The symptom of nasal discharge in this group was not as constant as in the previous group. An equal number complained of dryness as complained of increased discharge, and the nasal discharge complained of was of minor degree and did not compare to that of the first group.

Similarly, none in this group of patients had had any similar nasal complaints prior to the surgical interruption of the sympathetic fibers. Here, again, the symptoms followed the surgical procedure almost immediately and were present in about the same degree of severity

eighteen months to two years after the procedure. The patients in this group also noticed no change in their individual susceptibility to infections of the upper respiratory tract.

Nasal examinations revealed findings similar to those of the first group, with the exception that the nasal secretions were not noticeably increased in amount. The mucous membrane findings were similar, with marked swelling present in the membrane covering the septum and turbinate bodies. The color of the membrane varied from that of normal to slightly pale. The normal appreciation of pressure and pain remained unchanged, as did the sense of smell. No true polyps or polypoid degeneration was found. The response to the locally applied vasoconstrictor (0.25 per cent phenylephrine hydrochloride solution) was prompt and complete and again afforded temporary relief from the nasal symptoms. A biopsy specimen taken from the mucous membrane covering the middle turbinate body was again reported as presenting chronic inflammation of the mucous membrane of the upper respiratory tract. The microscopic section showed no abnormal change in the glandular elements.

A third group of patients studied consisted of 9 patients in which an anterior thoracic rhizotomy was performed in an effort to interrupt the preganglionic sympathetic efferents to the head. Postoperative study of the pupils and the sweating responses in these cases indicated that the sympathectomy was less complete than with either of the operations previously described. In this group one patient complained of nasal symptoms of stuffiness and excessive nasal mucoid discharge prior to operation. The remainder had no nasal complaints prior to the surgical procedure. The patient who had had symptoms prior to operation still had symptoms of nasal stuffiness postoperatively, but the nasal discharge was less in amount than he had had preoperatively. The remainder of this group all complained of nasal stuffiness of variable degree after the surgical procedure. Five had complaints of increased nasal discharge varying from serous to mucoid variety. The remaining 4 had no pronounced increase in the amount of nasal discharge, and 1 of this number had an actual decrease in the amount of nasal discharge as compared with the amount present preoperatively. Again the symptoms began almost immediately after operation, and although they were present after eighteen months to two years they were of a lower degree of severity, and, except for a few patients, the complaints of the patients were minimal. Here, again, there was no apparent change in the individual susceptibility to infections of the upper respiratory tract after the operation.

Nasal examinations revealed findings very similar to those of the preceding two groups, particularly in regard to the degree of swelling of the

mucous membranes. The amount of nasal secretions present varied in different persons from excess in amount to almost dryness of the membranes in 1 case. This case was the one in which there were complaints of excessive nasal discharge prior to the surgical procedure and a noticeable decrease in the nasal discharge after it.

There was no true polyp formation and no polypoid degeneration of the membranes. The appreciation of pressure and pain again remained unchanged in all cases, as did the sense of smell. The response to the locally applied vasoconstrictor (0.25 per cent phenylephrine hydrochloride solution) was again prompt and complete and afforded temporary relief from the nasal symptoms. We were not able to obtain a representative biopsy of nasal mucous membrane from this group of patients.

In 2 cases in which a left stellate ganglionectomy (preganglionic) and a right superior cervical ganglionectomy (postganglionic) were performed studies were made from the standpoint of the reaction of the nasal mucosa to locally applied epinephrine (adrenalin®) and to epinephrine administered systemically. A 1:1000 solution of epinephrine was applied locally to the mucous membrane bilaterally. The response to the vasoconstrictor was equal bilaterally, and the vasoconstrictor was moderately effective in relieving the symptoms of nasal obstruction. Similarly, 4 minims of a 1:1000 solution of epinephrine was injected intramuscularly, and the response of the nasal mucosa to vasoconstriction was observed to be about equal to that of the locally applied epinephrine solution.

Neither of the two methods produced as rapid or complete vasoconstriction as did locally applied 0.25 per cent phenylephrine hydrochloride solution. In 1 of the 2 cases mentioned observations were made on two different occasions. The first observation was made two weeks after the surgical procedure, and the second observation was made six months after the procedure with similar findings on both examinations. In the second case observations were made approximately one year after the surgical procedure.

Similar studies were made in 1 case of a unilateral stellate ganglionectomy, approximately nine months after the surgical procedure. Four minims of a 1:1000 solution of epinephrine injected intramuscularly produced an equal vasoconstrictor response in the nasal mucous membranes. Similarly, a locally applied solution of 1:1000 epinephrine produced an equal vasoconstrictor response of the nasal mucous membranes.

Although the mucous membranes on the sympathectomized side were more swollen than on the nonsympathectomized side, the vasoconstrictor response appeared to be equal. These observations suggest that, at least

so far as the nasal mucosa is concerned, a postganglionic sympathectomy does not increase the responsiveness to epinephrine, as described by White and Smithwick.[2]

The three operative procedures used in this series of cases vary as to the site at which the fibers of the sympathetic system were sectioned. The superior cervical ganglionectomy constitutes a postganglionic sympathectomy, whereas stellate ganglionectomy is a preganglionic sympathectomy. Both of these procedures result in complete sympathetic denervation of the nasal mucous membrane. The anterior thoracic rhizotomy also constitutes a preganglionic sympathectomy and, as performed in this series, it resulted in less complete sympathetic denervation of the cephalic areas. The vasoconstrictor fibers to the cephalic areas and to the nasal mucosa make their exit from the spinal cord in the upper thoracic segments and pass through the stellate ganglion as preganglionic nerves to the superior cervical ganglion. From this ganglion, postganglionic fibers are then distributed to the various cephalic areas.

As is evidenced by the nasal symptoms and physical findings in the mucous membrane of the nose found in the three groups of patients presented, the site of interruption of the sympathetic fibers to the nasal mucosa bears no striking relation to the type of symptoms produced but does bear a relation to the degree of symptoms produced. In all three groups essentially the same type of symptoms and physical findings were present, but in a variable degree. The symptoms of nasal stuffiness were constant in all three groups and, although variable in degree, were the chief presenting complaint in all cases.

The degree of swelling of the mucous membranes found on physical examination was equal in the first two groups but was of lesser degree in the third group of patients. The symptoms of nasal discharge varied considerably. Not only did the amount of nasal discharge vary in the individual groups but it also varied among the three groups. Nasal discharge was a more prominent symptom in those cases in which the superior cervical ganglion had been removed. In this group of 8 cases it was a fairly constant complaint.

In the remaining two groups the symptom of nasal discharge was of minimal degree and varied greatly from slight nasal discharge to even complaints of dryness. The mucous membranes showed no decided difference in appearance in the three groups except for the amount of moisture present in the nasal chamber. In no case was there found any great degree of dryness of the mucosa. In general, the symptoms and

2. White, J. C., and Smithwick, R. H.: The Autonomic Nervous System: Anatomy, Physiology and Surgical Application, ed. 2. New York, The Macmillan Company, 1941.

physical findings were severest in the cases of superior cervical ganglionectomy; they were less in degree in the cases of stellate ganglionectomy, and still less in degree in the cases of anterior thoracic rhizotomy.

A group of 8 patients observed immediately after a unilateral superior cervical ganglion block with a 1 per cent solution of procaine hydrochloride revealed nasal findings very similar to those previously described with the exception of the color of the mucous membrane. The nasal mucosa on the side of the block was of a greater degree of redness than the normal side and also showed a greater degree of swelling.

In contrast to the changes resulting from the interruption of the sympathetic supply to the mucous membrane of the nose, the opportunity presented itself to study the changes resulting from interruption of the parasympathetic supply. Six patients in whom the greater superficial petrosal nerve was sectioned for unilateral headache were available for observation. The section was unilateral in all cases, making possible the comparison with the side not denervated. The nasal findings in all cases were essentially the same except for minor structural variations.

The mucous membrane covering the septum and turbinate bodies showed much less swelling on the parasympathectomized side than on the normal side. The variation in the moisture of the membranes on the two sides was also striking. The parasympathectomized side showed a dryer mucosa with a moderate amount of crusting. The moisture content on the side not operated on was normal in amount. The color of the mucous membrane was the same on the two sides. Appreciation of pressure and pain remained unchanged, as did the sense of smell. The response to a locally applied vasoconstrictor (0.25 per cent phenylephrine hydrochloride) was equal bilaterally. The nasal symptoms complained of consisted of excessive dryness and crusting on the parasympathectomized side.

A biopsy specimen was obtained from the mucous membrane covering the middle turbinate body in a representative case. This specimen was reported as showing evidence of chronic inflammation and squamous metaplasia of the mucous membrane. The surface epithelium was of squamous or stratified squamous type. The mucous glands present showed moderate activity.

The physiologic changes that occur in the nasal mucosa in these patients can best be explained by understanding the nature of the vascular dynamics involved. The smooth muscle of the vascular tree is maintained in a normal state of tonicity by the antagonistic action of the parasympathetic and sympathetic systems. The physiology of its contraction involves not only a complex nutritional mechanism but a neuromuscular one as well. Sympathin is produced at the terminal endings of the sympathetic nerve fibers, while acetylcholine is produced at the terminal

fibers of the parasympathetic nerves and at the synaptic junctions of the sympathetic and parasympathetic systems. These chemical substances are concerned with the mechanism of smooth muscle contraction, and, normally, these two systems are balanced against each other, sympathin tending to cause contraction and acetylcholine tending to cause relaxation. To maintain this balance several enzyme systems are present. Adrenin or sympathin is destroyed by amine oxidase and acetylcholine is destroyed by choline esterase, both of which enzymes are present in the body tissues. The nasal congestion that results as a consequence of the interruption of the sympathetic impulses is due, in the main, to a loss of constrictor tone secondary to a decrease in the amount of adrenin or sympathin at the terminal sympathetic fibers necessary to maintain this tone. In addition, this vascular relaxation is further enhanced by the uninhibited activity of the vasodilator fibers of the parasympathetic system. The fact that the locally applied phenylephrine hydrochloride solution tends to overcome this congestion is further indication that the adrenin insufficiency is behind the loss of constrictor tone.

The increased secretory activity associated with our series of cases is due probably to the uninhibited parasympathetic activity. The activation of the glands of the mucous membrane of the nose is mediated solely through the parasympathetic system. In addition to the direct activation of the glands, the vasodilation present results in an increased capillary permeability, facilitating the discharge of serous fluids which provides a basis for increased secretory output of the glands.

Similarly, the changes in the mucous membrane of the nasal cavity resulting from interruption of the parasympathetic supply are due to changes in the vascular dynamics of the mucous membrane and to diminished activation of its glandular elements. In this instance there is an increased constrictor tone and a decrease in the activity of the vasodilator fibers, resulting in a state of vasoconstriction. The dryness of the mucosa is secondary to a decreased output of the glandular elements of the mucosa with a lack of adequate moisture content necessary for humidification of the inspired air. The decreased moisture content further results in interference with the normal ciliary activity, as a consequence of which the secretions that are present are not carried away in the proper manner but become dry and inspissated with the resultant formation of many cysts.

The symptoms and physical findings in this series of sympathectomized cases bear a striking similarity to those found in clinical cases of allergic rhinitis. This was observed by Fowler[3] in 1943, who described a case of unilateral vasomotor rhinitis and Horner's syndrome

3. Fowler, E. P., Jr.: Unilateral Vasomotor Rhinitis Due to Interference with Cervical Sympathetic System, Arch. Otolaryng. **37**:710 (May) 1943.

in a patient who had previously had a unilateral stellate ganglionectomy for causalgia of the median nerve. His patient presented much the same symptoms and physical findings that have been found in our series of cases. That the autonomic nervous system may play a part or an important role in allergic disease has been recognized previously. Kuntz [4] suggested that the common manifestations of allergy are associated with abnormal functional states of the autonomic nervous system and that not infrequently this modified functional status is an important factor in the cause of the so-called allergic state. The autonomic nervous system functions below the level of consciousness and serves to carry on the purely vegetative functions, such as glandular secretion, the action of smooth muscle and maintenance of vascular tone. These autonomic functions are significantly influenced and regulated by the diencephalic autonomic centers located in the hypothalamus. These central autonomic centers receive regulatory impulses from the cerebral cortex but are also capable of integrating autonomic reactions independent of these regulatory impulses. In most allergic diseases the functional disturbances encountered are related to an autonomic imbalance. The decreased vascular tone and the increased secretory activity in allergic rhinitis represents a decreased sympathetic and heightened parasympathetic activity. The heightened tonus of the bronchial musculature in bronchial asthma again represents an increased parasympathetic response. Thus the common manifestations of allergic disease are related to a heightened parasympathetic activity. Whereas the functional disturbances encountered in allergic disease are all apparently related to an autonomic imbalance, specifically a heightened parasympathetic response, this by no means should be interpreted as the site for development and production of allergic disease. The severest manifestations of allergic disease are, for the most part, associated with an antibody-allergen reaction in the tissue elements and are probably completely independent of the action of the autonomic system. That some intimate relation between the autonomic system and the production of allergic disease exists appears to be very probable. Exactly what this relation may be is as yet not known, and the problem remains to be investigated further.

SUMMARY

1. In a series of 27 cases in which the sympathetic nervous supply to the mucous membrane of the nose was interrupted surgically, symptoms of nasal stuffiness and nasal discharge resulted.

2. Swelling of the nasal mucosa covering both the septum and the turbinate bodies was associated with increased secretion.

4. Kuntz, A.: The Autonomic Nervous System, ed. 3, Philadelphia, Lea & Febiger, 1945.

3. The changes observed were the result of a loss of vasoconstrictor tone secondary to decreased formation of adrenin at the terminal endings of the sympathetic fibers and to uninhibited parasympathetic activity.

4. Postganglionic sympathectomy of nasal mucosa does not result in increased reactivity to epinephrine, whether applied locally or given intramuscularly.

5. In 6 cases in which the parasympathetic nerve supply to the nasal mucous membrane was interrupted the exact opposite in nasal symptoms and physical findings was found, that is, the mucous membranes were shrunken and dry.

6. The similarity of the appearance of nasal mucosa following sympathectomy to that of nasal allergy suggests that allergic disease and the autonomic system may be intimately associated in a manner not yet discovered.

7. The clinical application of sympathectomy and parasympathectomy of nasal mucosa is to be further investigated and is worthy of consideration.

HYALURONIDASE IN RHINOPLASTY

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DURING the last few years there has been a widespread increase in the clinical use of hyaluronidase, the specific enzyme now generally conceded to be the "spreading factor" of Duran-Reynals [1] and McClean.[2] Various authors have reported its effectiveness in facilitating the absorption of fluids by hypodermoclysis [3] and in effecting better local anesthesia in general surgery and dentistry [4] and its effectiveness in ophthalmology [5] and other varied clinical applications.[6] We therefore decided to explore its possibilities in the field of rhinoplastic surgery.

Hyaluronic acid, a chemical gel, functions in tissues of the body as a cement substance between cells, thus acting as a barrier to the diffusion of tissue fluids. Hyaluronidase is the specific enzyme which

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1. Duran-Reynals, F.: The Effect of Extracts of Certain Organs from Normal and Immunized Animals on the Infecting Power of Vaccine Virus, J. Exper. Med. **50**:327 (Sept.) 1929.

2. McClean, D.: The Influence of Testicular Extracts on Dermal Permeability and Response to Vaccine Virus, J. Path. & Bact. **33**:1045 (Oct.) 1930.

3. (*a*) Hechter, O.; Dopkeen, S. K., and Yudell, M. H.: The Clinical Use of Hyaluronidase in Hypodermoclysis, J. Pediat. **30**:645 (June) 1947. (*b*) Schwartzman, J.; Henderson, A. T., and King, W. E.: Hyaluronidase in Fluid Administration, ibid. **33**:267 (Sept.) 1949.

4. (*a*) Kirby, D. K.; Eckenhoff, K. E., and Looby, J. P.: Use of Hyaluronidase with Local Anesthetic Agents in Nerve Block and Infiltration Anesthesia, Surgery **25**:101 (Jan.) 1949. (*b*) Looby, J. P., and Kirby, C. K.: Use of Hyaluronidase with Local Anesthetic Agents, J. Am. Dent. A. **38**:1 (Jan.) 1949.

5. Atkinson, W. S.: Use of Hyaluronidase with Local Anesthesia in Ophthalmology, Arch. Ophth. **42**:628 (Nov.) 1949.

6. Som, M. L.; Schneierson, S. S., and Sussman, M. L.: Enhancement of Penetration of Penicillin into Inflamed and Normal Mucous Membrane by Hyaluronidase, Proc. Soc. Exper. Biol. & Med. **70**:96 (Jan.) 1949. Simon, N., and Narins, L.: Effect of Hyaluronidase on the Absorption of a Subcutaneously Deposited Radiopaque Substance, Am. J. Roentgenol. **61**:91 (Jan.) 1949.

hydrolyzes and depolymerizes hyaluronic acid,[7] with the resultant lessening of the viscidity of the gel and consequent facilitation of diffusion of tissue fluids. The rate of diffusion is proportionate to the amount of enzyme available, and the extent of effectiveness is proportionate to the volume of solution present[8]; in addition, local interstitial pressure and mild local massage further enhance the rapidity of action of the hyaluronidase.[8a] The early reports of the use of testicular extract (which was the first source of the spreading factor) for increasing the diffusion of fluids were accepted with mixed reactions because the stimulation of cell growth in vitro by this extract suggested[9] the possibility of undesirable side effects and discouraged its clinical use.

However, further comprehensive studies[10] have shown the absence of harmful effects in therapeutic doses and the practically complete absence of local and general toxic and allergic reactions. With its introduction into clinical use for local anesthesia, Kirby, Eckenhoff and Looby[4a] noted that hyaluronidase decreases the time of effectiveness of the local anesthetic but that the addition of 1:1,000 epinephrine hydrochloride to the 1 per cent procaine hyaluronidase solution restored the duration of the anesthesia.

In our study, 50 routine consecutive patients underwent rhinoplastic procedures, often in combination with submucous resection of the septum. They were observed postoperatively from day to day and then followed after discharge from the hospital. For local anesthesia of the external nose a solution of 125 units of hyaluronidase to 25 cc. of 1 per cent procaine with 0.5 cc. of 1:1,000 solution of epinephrine hydrochloride was used. Beginning intranasally in the intercartilaginous area just cephalic to the plica vestibuli, 3 cc. of the solution was injected along each side of the nose upward to the frontal bone medial to the inner canthus of the eye to anesthetize areas supplied by the infratrochlear nerve and the external nasal branch of the anterior ethmoidal nerve. Next, 1 cc. was injected around the area where the infraorbital branch of the maxillary nerve emerges from its foramen; 1 cc., just below the inner canthus and 1 cc., below the foramen lateral to the alar-facial fold. Then, 0.5 cc. of the solution was injected in the nasal mucosa around the incisor canal approximately 2 cm. from the columella to anesthetize

7. Meyer, K.: The Biological Significance of Hyaluronic Acid and Hyaluronidase, Physiol. Rev. **27**:335 (July) 1947.

8. (*a*) Hechter, O.: Studies on Spreading Factors: Importance of Mechanical Factors in Hyaluronidase Action in Skin, J. Exper. Med. **85**:77 (Jan.) 1947. (*b*) Hechter, Dopkeen and Yudell.[3a]

9. Hoffman, D. C.; Parker, F., Jr., and Walker, T. T.: Effect of Testicle Extract on Rous Sarcoma, Am. J. Path. **7**:523 (Sept.) 1931.

10. Burket, L. C. and György, P.: Clinical Observations on the Use of Hyaluronidase, Pediat. **34**:56 (Jan.) 1949. Hechter, Dopkeen and Yudell.[3a] Schwartzman, Henderson and King.[3b]

the nasopalatine nerve. Lastly, the membranous septum and each ala were well infiltrated.

In order to study the effectiveness of the hyaluronidase solution, 20 patients were given injections of the prepared solution into one side of the nose and of the usual procaine-epinephrine hydrochloride solution into the other side. Increased thickening of the skin over the bridge of the nose toward the inner canthus of the eye and the thickening and doughy consistency of the alae were noted when the spreading factor was not used, whereas the side of the nose which was infiltrated with anesthetic containing hyaluronidase was remarkably free from these distortions within thirty seconds.

During operations, the areas over the superior maxillas where sawcuts were to be made and the regions about the lower lateral cartilages and the alae were frequently reinfiltrated to reenforce the anesthesia. Other areas, especially over the dorsum, became sensitive again after the operation had lasted for an hour or more, and such areas also were often reinfiltrated. Again it was startling to note the rapidity of dispersion of the injected fluid. Most remarkable was the almost immediate alleviation of pain and all sensitivity.

Any painful area about the nose or septum when infiltrated with the solution becomes insensitive within thirty seconds. This was particularly noteworthy in recent fractures of the nose and septum. In 12 patients with varying amounts of swelling and distortion, the usual procaine injection was followed by limited degrees of anesthesia, and in at least half manipulation was not feasible because of pain on pressure on the nose. After an additional injection of the hyaluronidase-procaine-epinephrine hydrochloride solution, the swelling of the original inflammation following injury, plus that produced by the first procaine injection, became markedly diminished and the anesthesia became complete. Accurate diagnosis of bone and cartilage deformities was facilitated because of the lessened distortion of the soft tissues, and the replacement and reposition of the hard parts were made easier.

In many of our cases operation lasted about three hours, and with some reinjection of solution at the halfway mark and occasional small regional injections at the end of the operation, these patients were carried through the long surgical procedures with completely satisfactory anesthesia. When anesthesia with the usual procaine-epinephrine hydrochloride solution was administered, the final modeling of the nose was more or less painful for some patients. In these a moderate injection of hyaluronidase-procaine-epinephrine hydrochloride has produced immediate and complete desensitization without the disadvantage of distorting the soft tissues, which at this crucial point in the operation could block the adequate appraisal of the results achieved.

We have a definite opinion that hemostasis is certainly not made worse; on the contrary, as far as our clinical observations permit, we have concluded that there is less bleeding than when the spreading factor is not used. Furthermore, it is our impression that postoperative edema and ecchymoses are less frequent than heretofore and also that the duration of the postoperative sequelae is noteworthily diminished. In a great measure, the latter is to be expected, because if at the end of the operation there are a minimum of swelling of the soft tissues and less subcutaneous secretion, the adjustment of a well fitting dressing is more possible. Organization of the diminished amount of subcutaneous fluids results in less scar tissue beneath the skin and better final postoperative results are to be expected. We have not seen any systemic or local complications which could be attributed to the use of the enzyme.

The following advantages of the use of hyaluronidase-procaine-epinephrine hydrochloride solution in rhinoplastic surgery may be listed: (1) prompt subsidence of distortion of tissues, important in deciding on the amount of plastic correction produced and in evaluating the dimensions and shape of the columella, alae and lower lateral cartilages and their relation to the proper functioning of the nose; (2) more adequate and complete anesthesia due to better diffusion of the procaine into the tissue; (3) more rapid onset of anesthesia (practically instantaneous), thus saving operating time, and (4) most effective supplementary anesthesia.

MÉNIÈRE'S SYNDROME

A Proposed Classification and Differential Diagnosis

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IN REVIEWING the literature on the subject of Ménière's syndrome, one is amazed at the confusion which exists regarding the nomenclature and etiology of the symptom complex of vertigo, tinnitus and impaired hearing.

What one author calls Ménière's syndrome, another calls Ménière's disease, and still others will list this symptom complex as aural vertigo or give it other names.

Politzer [1] applied the term Ménière's disease to the acute explosive type or apoplectiform attack irrespective of etiology. Ménière's symptom complex he considered as cases of vertigo, tinnitus and deafness occurring in patients with previous aural disease. Later, Politzer applied the term Ménière's disease to various diseases of the ear and central nervous system, manifested by attacks of dizziness.

Furstenberg [2] applied this term to the explosive, vertiginous attack which fells the victim in his track and which is the result of interruption of the circulation to the brain and retention of sodium ion by the body, suggesting an increased avidity or unusual sensitivity to sodium ion of the local tissues involved.

Lindsay [3] applied the term Ménière's disease to the idiopathic type only in which the causation, pathogenesis and reason for the clinical disturbances are not known and which is characterized by recurring attacks of vertigo associated with auditory disturbances of a fluctuating character. The symptoms are due to hydrops of the labyrinth and stasis

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1. Cited by Fisher, J., and Wolfson, L. E.: The Inner Ear, New York, Grune & Stratton, Inc., 1943.

2. Furstenberg, A. C.: Summation: Including Otoneurology, Otosurgery, and Problems in Modern Warfare, Symposium on Vertigo, Ann. Otol., Rhin. & Laryng. **56**:576 (Sept.) 1947.

3. Lindsay, J. R.: Pathology of Vertigo Arising from the Peripheral Vestibular Apparatus, Ann. Otol., Rhin. & Laryng. **56**:541 (Sept.) 1947; Labyrinthine Dropsy and Ménière's Disease, Arch. Otolaryng. **35**:853 (June) 1942.

of the endolymph with distention of the membranous labyrinth and herniation of the utricle and saccule into the semicircular canals as the result of hypersecretion or lack of absorption of endolymph, or both.

In one description Ménière [4] himself called the attack Ménière's disease and attributed it to the hemorrhagic plastic exudate in the semicircular canals.

Brunner [1] contended that the vasomotor disturbances producing exudation of plasma and cells in the inner ear or labyrinth—the idiopathic Ménière's syndrome, so-called vasomotor otitis interna—are characterized by intermittent attacks of short duration, often with simultaneous occurrence of migraine, angioneurotic edema and vasomotor rhinitis.

Kobrak [1] called the condition angiopathica labyrinthica, indicating the causes as vascular in origin.

Crowe [5] stated the belief that Ménière's syndrome is a disorder of the normal static pressure and of the chemical constituents of the endolymph.

Dandy [6] expressed the opinion that the lesion in Ménière's syndrome is in the auditory nerve.

Wright [7] stated the belief that it was a focal labyrinthitis or bacterial intoxication.

Mygind and Dederling [8] assumed that vasomotor disturbances were causative factors leading to a retention of water generally as well as in the inner ear in Ménière's syndrome.

Lochman,[9] of the Hadassah Rothschild Hospital, in Jerusalem, stated that the syndrome may be due to both vasoconstriction and vasodilatation as a result of low histamine tolerance and allergic stimulation.

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4. Williams, H. L.: A Review of Ménière's Original Papers in the Light of Our Present Knowledge of Ménière's Disease, Ann. Otol., Rhin. & Laryng. **58**:761 (Sept.) 1949.

5. Crowe, S. J.: Ménière's Disease: Study Based on Examinations Made Before and After an Intracranial Division of the Vestibular Nerve, Medicine **17**:1 (Feb.) 1938.

6. Dandy, W. E.: Pathological Changes in Ménière's Disease, J. A. M. A. **108**:931 (March 20) 1937.

7. Wright, A. S.: Further Clinical Observations on Nature and Treatment of Ménière's Disease, J. Laryng. & Otol. **55**:383 (Aug.) 1949.

8. Mygind, S. T., and Dederling, D.: Ménière's Disease as an Indicator of Disturbances in the Water Metabolism, Capillary Function, and Body Conditions, Ann. Otol., Rhin. & Laryng. **55**:61 (March) 1938.

9. Lochman, J. (Hadassah-Rothschild Hospital, Jerusalem): Personal communications to the author

Shelden and Horton [10] expounded the theory of increased permeability of the capillaries, endolymph stasis and resultant hydrops of the labyrinth, allergic in origin.

Atkinson [11] classified Ménière's syndrome as the allergic type, in which the histamine test is positive and which responds to histamine desensitization, and as a vasospastic type, which responds to thiamine and nicotinic acid.

Witmaak [12] found in 2 cases obstruction in the cochlear aqueduct by concretions.

Another confusing term used is pseudo-Ménière's syndrome. Hochwarth [1] applied the term to paroxysmal attacks of vertigo, tinnitus and deafness occurring in patients who never had any aural disease. Hewson applied it to the condition caused by chemical or toxic changes which responded to a salt free diet. Dandy [6] considered pseudo-Ménière's syndrome as an attack of vertigo without tinnitus and deafness. Lochman [9] considered it an attack in which two out of the three symptoms (vertigo, tinnitus, deafness) were present. Lindsay reserved this term for vertigo without auditory disturbances or symptoms referable to the central nervous system.

The commonest and most annoying symptom in Ménière's syndrome is vertigo. Vertigo may originate in the inner ear, in the vestibular nerve, in the cerebellopontile angle, in the brain stem, cerebellum and cerebral cortex.[13] Vertigo may occur in functional diseases, such as anxiety, hysteria, neurasthenia, neurosis, psychosis, hypochondria and malingering.[13]

In view of the different sites of origin of the triad of symptoms of vertigo, tinnitus and deafness, which may occur singly or in combination, and in view of the different etiology, it is proposed that Ménière's syndrome be classified as peripheral, central or psychosomatic.

The term Ménière's disease should be dropped, since it does not constitute a definite pathologic entity.

The Ménière syndrome of peripheral origin can further be classified as follows: (1) acute; (2) subacute and chronic; (3) idiopathic, and (4) pseudo-Ménière's.

The acute type is explosive in onset. Its causes are allergy, hemorrhage, thrombosis, embolism into the internal auditory artery and its

10. Shelden, C. H., and Horton, B. T.: Treatment of Ménière's Disease with Histamine Administered Intravenously, Proc. Staff Meet., Mayo Clin. **15**:17 (Jan.) 1940.

11. Atkinson, M.: Ménière's Syndrome: The Basic Fault, Arch. Otolaryng. **41**:385 (Oct.) 1946; Vertigo, Canad. M. A. J. **42**:326 (April) 1940.

12. Witmaak, cited by Dandy.[6]

13. Levy, L., and O'Leary, J. L.: Incidence of Vertigo in Neurological Conditions, Ann. Otol., Rhin. & Laryng. **56**:557 (Sept.) 1947.

terminal branches, leukemia, infectious mononucleosis, rapid descent in an airplane, caisson disease, transverse fracture and longitudinal fracture of the labyrinth, concussion, blast, serous labyrinthitis, suppurative labyrinthitis and toxic and febrile states.

The signs and symptoms are explosive in onset. Severe vertigo and nystagmus, past pointing, falling, pallor, perspiration, nausea, vomiting, tinnitus and impaired hearing to total deafness. The attack may last from a few hours to five to six weeks. The signs and symptoms are combined and proportionately increased or decreased.

In the subacute and chronic type the irritant is milder and of slow gradual onset. Its causes are adhesive tubotympanic disease,[14] toxic labyrinthitis, neurolabyrinthitis, otosclerosis and ill effects of streptomycin, quinine, morphine, salicylates and other drugs.

The signs and symptoms are vertigo, tinnitus and deafness, partial or complete, of the conduction type in early cases, mixed in late cases. Spontaneous nystagmus may or may not be present; it is usually absent.

The idiopathic condition and the pseudo-Ménière syndrome were previously discussed.

Ménière's syndrome of central origin can be caused by vascular, inflammatory and space-taking lesions. The signs and symptoms may occur singly or in combination and are usually disproportionate spontaneously or on vestibular stimulation.

In Ménière's syndrome of psychosomatic origin the signs and symptoms are inconsistent, variable and influenced by suggestion. There is a psychosomatic background.

According to Spiegel,[15] the automatic nervous system may affect the vestibular mechanism by vasomotor disturbance in the distribution of the internal auditory artery in the inner ear, eighth nerve nuclei in the brain stem, in migraine vasoconstriction spasm and in variations of blood pressure, in which case the pulse wave affects the endolymph, causing vertigo. The autonomic nervous system[16] is contained in the splanchnic nerves and vagi, along which impulses travel to the vestibular mechanism and from the vestibular mechanism to the autonomic nervous system, and is very readily influenced by psychologic stimuli. The autonomic center lies close to the vestibular nuclei in the brain stem; the vestibular nuclei capillaries may become increasingly sensitive as a result of allergic reaction and may be the cause of vertigo and influence the autonomic nervous system.

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14. Meriča, F. W.: Vertigo Due to Obstruction of Eustachian Tubes: Clinical Study Based on 135 Cases, J. A. M. A. **118**:1282 (April 11) 1942.

15. Spiegel, E. A., and Sommer, I.: Neurology of the Eye, Ear, Nose, and Throat, New York, Grune & Stratton, Inc., 1944, p. 139.

16. Wechsler, I. S.: The Autonomic Nervous System, in a Textbook of Clinical Neurology, ed. 5, Philadelphia, W. B. Saunders Company, 1944, pp. 668-678.

Emotion may produce, modify and influence physical disease,[17] a fact established physically, biochemically and psychologically, some examples of which are asthma, hypertensive vascular disease, migraine, postnasal drip, congestion and secretion of the eustachian tube and change of p_H from acid to alkaline in nasal secretion.

The central nervous system, the vestibular system and the autonomic nervous system are integrated and are easily affected by psychic influences.

DIFFERENTIAL DIAGNOSIS

Nystagmus.—In a peripheral lesion nystagmus is usually a mixed nystagmus; horizontal-rotatory may be pure horizontal or rotatory of the same intensity and frequency. It may be of the first, second or third degree.

In a central lesion the spontaneous and induced nystagmus may be vertical, oblique, perverted or inverted, pure horizontal or pure rotatory of different amplitude and frequency.

In a psychosomatic lesion there may be a convergence nystagmus, vertical end position nystagmus or dissociated fatigue nystagmus.

Vertigo.—Vertigo may be turning, tactile, lateral pulsion. The turning type of vertigo, the rotatory, is made worse on extreme lateral gaze in direction of the quick component of nystagmus, and the subject lies on the affected side. In tactile vertigo the subject has a sensation of falling out of a chair or bed. In lateral pulsion vertigo the subject has a feeling of being pulled or pushed to either side.

In a peripheral lesion the vertigo originates in the inner ear or labyrinth. It is usually turning or tactile. It is toward the quick component of nystagmus and associated with falling and past pointing, proportionately. It may last from a few hours to five to six weeks. Vestibular tests show proportionate increase or decrease in vestibular responses in many instances. The onset is sudden and violent in acute cases, moderate to mild in subacute and chronic cases.

In a central lesion the vertigo originates in the medulla, pons, cerebellum, cerebrum and in their pathways. Increased intracranial pressure may be present. The onset of vertigo is slow and progressive in tumors and explosive in vascular and inflammatory lesions. It is not necessarily associated with nystagmus, falling and past pointing and is disproportionate spontaneously and on vestibular tests. The vertigo is usually progressive and may last for months and years. It may be rotatory, tactile and lateral pulsion.

17. Weiss, E., and English, O. S.: Psychosomatic Medicine: The Clinical Application of Psychopathology to General Medical Problems, Philadelphia, W. B. Saunders Company, 1943.

In a psychosomatic lesion the vertigo is functional in origin, may be mild turning or tactile; oftenest it is a sense of uncertainty. It is brought on by ocular fatigue and psychic strain, is inconsistent and variable in direction. It may follow deep breathing and respiratory arrhythmia. There is a vasomotor disturbance, a congenital or acquired excitability and a psychosomatic personality. Vestibular tests are apt to show hyperactive responses unless imposed on a degenerative organic lesion.

Post-traumatic vertigo[18] may last a long time following brain concussion, contusion or laceration. It may be associated with headache, an emotional state, fatigue, impaired memory, poor concentration, tinnitus, impaired hearing.

Vestibular tests will disclose the site of damage (peripheral or central) responsible for the vertigo.

Falling.—In a peripheral lesion falling is usually associated with nystagmus, vertigo, past pointing. Nystagmus is usually of second and third degree. Falling is toward the slow component. The direction of falling is changed on change of position of the head. There is a protective attempt against injury in falling. The spontaneous and induced phenomena are usually proportionate.

In a central lesion falling may occur singly or in combination with nystagmus, vertigo, past pointing. It is independent of direction of nystagmus and change of position of the head. Falling is to the affected side in cerebellar lesions and to the opposite side in cerebral lesions. There is no protective attempt against injury in falling. The spontaneous and induced phenomena are disproportionate.

In a psychosomatic lesion there may be a mere swaying or astasia-abasia. The falling is inconsistent, with no definite direction, and influenced by suggestion. There is a psychosomatic background. The subject makes no attempt to counteract or to maintain balance but manages to land in a soft spot just the same.

Past Pointing.—In a peripheral lesion absence of past pointing with both arms, after stimulation in the presence of a spontaneous past pointing toward the diseased side, suggests unilateral disease of the labyrinth or vestibular nerve and may be the first sign in neurolabyrinthitis of syphilitic origin.

In a central lesion there is an absence of past pointing in the homolateral arm on vestibular stimulation in the presence of spontaneous past pointing of the homolateral arm toward the diseased side. If past pointing does not improve on repetition of tests, it is pathologic.

18. Brown, H. A.: Posttraumatic Syndrome of Head Injury, Ann. Otol. Rhin. & Laryng. **50**:1152 (Dec.) 1941.

In a psychosomatic lesion past pointing may occur in connection with vertigo in hypnosis as a result of suggesting of rotational vertigo. It occurs inconsistently and does not fit in with other labyrinthine or neurologic findings.

Tinnitus and Impairment of Hearing.—In a peripheral lesion there are tinnitus and impairment of hearing of the conduction type (later, also of the nerve or perceptive type) with atrophy of the organ of Corti and ganglionic cells.

In a central lesion deafness varies from nerve or perceptive type of partial deafness in temporal lobe lesions to complete deafness in acoustic neurinoma.

In a psychosomatic lesion there are tinnitus and hypersensitivity to acoustic stimuli, which are variable and inconsistent.

Last but not least in importance are the vestibular tests in the differential diagnosis: the Bárány tests (which are the standard tests and the most reliable), the mass caloric and the turning tests. The minimal tests are the Kobrak test and the modification of the Kobrak test, which have many disadvantages and are by no means so reliable as the Bárány tests. There are also the galvanic tests, which are useful only in cases in which the caloric tests cannot be used.

SUMMARY

It is proposed that Ménière's syndrome be classified as of peripheral, central or psychosomatic origin.

The term pseudo-Ménière is synonymous with vertigo and does not mean anything else.

The term Ménière's disease should be dropped in favor of that of Ménière's syndrome, to eliminate the present confusion regarding the difference in the nomenclature.

The principal differential diagnostic points between a peripheral and central lesion are the periodicity of attacks, freedom from symptoms between attacks and the presence of cochlear symptoms such as tinnitus, and fluctuating hearing impairment (conduction type first and later mixed conduction and perceptive type), in addition to the combined proportionate association of vertigo, past pointing, nystagmus, falling in the acute attacks, presence of vestibular test findings suggestive of a peripheral lesion and absence of positive neurologic signs and symptoms.

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SYNDROME OF BILATERAL VESTIBULAR PARALYSIS AND ITS OCCURRENCE FROM STREPTOMYCIN THERAPY

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THE DISTURBANCE of equilibrium caused by bilateral loss of vestibular function from streptomycin therapy appears to be basically similar to that of bilateral vestibular paralysis from other causes, whether the site of the lesion is in the peripheral or the central components of the vestibular nervous system. The syndrome of bilateral vestibular paralysis has rarely been noted in the literature, and not before its happening as an untoward effect of streptomycin therapy has there been an opportunity to observe a considerable number of patients with this syndrome from its onset and through the stages of its regression. The practice of prescribing smaller doses of streptomycin than formerly and of substituting other antibiotics with less neurotoxic effects makes the study of the symptomatology of the toxicity of streptomycin, including its pathologic foci, of importance chiefly in obtaining a better understanding of disorders of the function of the acoustic nerve as an aid in diagnosis. Bilateral vestibular paralysis may occur from causes other than streptomycin, and disturbance of equilibrium not due to that drug may be experienced by patients receiving streptomycin. Six of the patients observed in this series of 56 had disturbances of gait which were not caused by the toxicity of streptomycin. The diagnosis in those cases were meningoencephalitis (1); peripheral neuritis (1); hysteria (1), and sequela of tuberculous meningitis (3).

Continued observations on patients who received streptomycin necessitate some modification of the symptomatology as previously reported,[1] but the information is chiefly additive rather than contradictory and,

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1. Northington, P.: Effects of Streptomycin on Eighth Cranial Nerve Function, read before the Otolaryngologic Scientific Session at the Thirty-Fourth Clinical Congress of the American College of Surgeons, Los Angeles, Calif., Oct. 20, 1948.

along with other reports,[2] is in agreement generally on the diagnostic symptoms. The common pattern observed is as follows: A few hours after the appearance of one or more of the complaints, namely, dizziness on movement in bed, sensation of fulness in the head, noises in the ears, headache, nausea and vomiting, the patients experienced inability to maintain customary posture and gait, along with jumbling of distant visual objects, all the complaints increasing in severity, but usually for only a few days. A regression from this peak occurred, even when streptomycin therapy was continued, in which some amelioration of intensity of symptoms took place over a few days and then was usually followed by several months of slow and gradual improvement in posture, gait and vision and diminution of vertigo.

Impairment of hearing, which was perceptive in type, was infrequent, and in those so affected the tinnitus seemed to be constant and intense. In only 2 of the 6 in this series of 56 patients who were considered to have a hearing loss from streptomycin therapy was the impairment sufficient for them to be aware of it.

The headache was of a generalized type and usually subsided before the end of the acute stage; but in some cases it lasted several months, occurring in periodic attacks, and little relief was obtained from sedative drugs.

The tinnitus, a very common symptom, and usually the earliest, was described variously as a humming, buzzing, crackling, thumping or motorboat sound; the sensation was not always limited to the ears, but in some patients extended to the whole head. The tinnitus was intermittent and subsided soon in most of the patients who did not acquire any impairment of hearing. In some patients, however, tinnitus was especially annoying and was described as intensified or "set off" by any loud extraneous noise. A few patients complained of transitory tingling or numbness of the face, particularly about the mouth. The sensory disturbances of the head were variously described and not always easily classified. As an example, an old Negro said, "My head began to roar

2. Brown, H. A., and Hinslaw, H. C.: Toxic Reactions of Streptomycin on the Eighth Nerve Apparatus, Proc. Staff Meet., Mayo Clin. **21**:347 (Sept. 4) 1946. Farrington, R. F.; Smith, H. H.; Bunn, P. A., and McDermott, W. L.: Streptomycin Toxicity: Reactions to Highly Purified Drug on Long-Continued Administration to Human Subjects, J. A. M. A. **134**:679 (June 21) 1947. Fowler, E. P., Jr., and Seligman, E. L.: Otitic Complications of Streptomycin Therapy, ibid. **133**:87 (Jan. 11) 1947. Glorig, A., and Fowler, E. P., Jr.: Tests for Labyrinth Function Following Streptomycin Therapy, Ann. Otol., Rhin. & Laryng. **56**:379 (June) 1947. Snell, F. B.: Otitic Complications of Streptomycin Therapy, Mil. Surgeon **102**:202 (March) 1948. Winston, J.: Vestibular Findings Before, During and After Vestibular Damage Resulting from Streptomycin Therapy, Arch. Otolaryng. **77**:746 (June) 1948. Wallner, L. J.: The Otologic Effects of Streptomycin Therapy, Ann. Otol., Rhin. & Laryng. **58**:111 (March) 1949.

like a lion's den, and then it took off like a balloon, and I didn't know where my head was until I grabbed it between my hands."

Although neither nausea nor vomiting was common, even in the acute stages, and these symptoms did not appear to be related to the degree of vertigo, in some patients they were so distressing as to be the reason for discontinuing streptomycin therapy soon after it was begun.

Spontaneous nystagmus was not seen with the eyes in primary position, and when seen on rare occasions it was on lateral fixation and was of horizontal-rotary type.

The usual course in which the initial symptom of headache, aural noises, nausea or vomiting subsided soon after their onset was in contrast to that of the slow regression of the symptoms of vertigo, disturbance of gait and posture and the jumbling of visual objects.

The vertigo was not similar to the customary rotary type associated with nystagmus and past pointing that one is accustomed to find in acute unilateral vestibular disorders, in which the past pointing, or what may be termed forced movements, is a voluntary action augmented by tonic reflexes in a direction opposite that of the subjective vertigo, as if to maintain orientation or balance. The sense of false motion complained of by the patients who had received streptomycin was described as a feeling of being compelled to continue the movement of the body in the direction in which the head was tilted, chiefly to the face up or the face down position, as well as the sensation of continuing on the course in which the subject was walking after turning the head in a different direction. In the chronic stage, when improvement in gait was considerable, the patient was likely to bump a nearby object in passing, having a sensation of being drawn to it, a tendency which was increased on his giving thought to avoiding it.

The attempt of these patients to maintain the customary standing posture and gaits has such a close resemblance to the disturbance of equilibrium brought on by excessive use of alcoholic beverages that both patients and others noticed it. Not uncommonly, patients compared their disability from day to day to that produced by a certain number of "drinks." In the acute stage, the patients with the greatest disability were unable to stand or walk unsupported; in patients less severely affected, the ability to maintain a standing position was faulty, especially when the body was tilted in any direction. When the patient walked with a wide base with the body tilted slightly forward, he veered to one side or the other and frequently would have fallen without the aid of some support. Later, with regression in the severity of the symptoms, this disturbance in gait was more clearly noted, especially when the patient was walking on an uneven surface, and when going up or down grade this disturbance of equilibrium was increased. When the patient was

able to walk straight away with good ability, if he turned his head abruptly to change his course, he would not execute the maneuver with precision.

Superficial and deep reflexes were normal throughout the duration of observation, and there was no evidence of muscular weakness or spasticity.

The course of jumbling of visual objects generally paralleled the changing degree of disturbance of gait. Although patients usually read newspapers or books with ease, objects viewed beyond a few feet appeared to "run together" and lose detail on movement of the head; especially was this experienced on walking or riding in an automobile over a rough road. It was observed equally well with monocular and with binocular vision. In none of the patients on whom an ophthalmologic examination was made at the time of this complaint was any abnormality found, whether of visual acuity, fields of vision, muscular balance or accommodation or of the fundus.

The disorder of vision, posture and gait, along with vertigo, was regularly accompanied with a bilateral loss of vestibular reactions to both rotatory and caloric tests, which was total in some patients, and the degree of response was usually, but not always, proportional to that of the clinical disability. In only 1 case was there a detectable difference in degree of the reactions of the right and the left ear, and the clinical symptoms presented by this patient were similar to those of persons whose impairment of vestibular reactions were of equal degree on the two sides.

It is noteworthy that the responses on vestibular testing when present were always normal in type and more active usually in the caloric tests than in the rotatory tests, and the intensity of the symptoms in either the acute or the chronic stage was not consistently proportional with the degree of reaction in vestibular tests.

In several patients on whom galvanic testing (hand to ear) was done, the degree of reactions closely paralleled that found on the caloric test. Normal responses were obtained in the several patients on whom optokinetic nystagmus tests were made.

In none of the patients, four years being the longest any one has been observed, who had more than minimal symptoms of bilateral vestibular paralysis in the acute stage has there been either complete recovery on clinical examination or normal finding in vestibular tests. In those with impairment of cochlear function the hearing loss has not been restored. A few patients with the gross disturbance of vision and gait of the type characteristic of bilateral vestibular paralysis have recovered from their disability to the excellent degree of compensation found in the chronic stage following bilateral ablation of vestibular receptors. Although this status may be considered by some as complete

recovery, the residual disturbance of equilibrium is lasting for precision in locomotion in the dark and may be demonstrated easily and is, in fact, frequently made obvious to the patient, particularly if one attempts to swim or dive without the aid of vision. It is highly advisable, therefore, to warn those with bilateral vestibular paralysis who have recovered even to the extent of being able to engage in ordinary activities to avoid situations in which loss of equilibrium would be hazardous.

In 1882, James [3] published an account of his investigations of susceptibility of deaf-mutes to dizziness, recording that some were immune to being made dizzy by whirling in a swing and that, in general, this group of persons had difficulty in walking without the aid of vision and were disoriented when they were below the surface of water. James reasoned that those immune to dizziness would not suffer from seasickness, and he made the novel suggestion that in the treatment for seasickness blisters be used behind the ears as a counterirritant to the excitement of the organs beneath. I have found only the visual disorder common in bilateral vestibular paralysis to be recorded within recent years. Dandy [4] and Williams [5] wrote of the visual disorder and disturbance of gait following their treatment of Ménière's disease. Symptoms comparable to those of bilateral vestibular paralysis from streptomycin therapy occurred in Dandy's patient after division of both vestibular nerves, and in Williams' patient they followed bilateral labyrinthotomy. Neither author mentioned the permanent disorder of equilibrium that results from bilateral destruction of the vestibular receptors. Levin [6] reported the disturbance of posture, particularly in water, staggering in the dark and the difficulty of maintaining ocular fixation when the head is moved in a patient with bilateral vestibular paralysis of unknown origin. In addition, in 4 cases reported by Chusid and de Gutiérrez-Mahoney [7] the symptoms of bilateral vestibular paralysis were basically similar to those produced by toxicity to streptomycin therapy. The cause in those 4 cases was ascribed to scrub typhus, scarlet fever, syphilis and in 1, although the cause was obscure, the probable cause was reported to be scrub typhus.

A recent case of bilateral injury to the acoustic nerve is reported.

A sailor aged 37 was transferred to this hospital, with the complaint of bilateral deafness and disorder of gait. Three months previously he had fallen a distance of

3. James W.: The Sense of Dizziness in Deaf-Mutes, Am. J. Otol. **4**:239 (Oct.) 1882.

4. Dandy, W. E.: The Surgical Treatment of Ménière's Disease, Surg., Gynec. & Obst. **72**:421 (Feb. no. 2 A) 1941.

5. Williams, H. L.: The Present Status of the Diagnosis and Treatment of Endolymphatic Hydrops (Ménière's Disease), Ann. Otol., Rhin. & Laryng. **56**:614 (Sept.) 1947.

6. Levin, P. M.: The Syndrome of Vestibular Paralysis in Man, J. Nerv. & Ment. Dis. **89**:335 (March) 1939.

7. Chusid, J. G., and de Gutiérrez-Mahoney, C. G.: The Syndrome of Bilateral Vestibular Paralysis, J. Nerv. & Ment. Dis. **103**:172 (Feb.) 1946.

about 15 feet (4.5 meters), striking his head on a boom in the water. On examination, twenty-four hours after the accident, according to his health record, only pain in back of the head, deafness, disturbance of equilibrium and paralysis of the left side of the face were noted. When seen by me three months later, he had no complaint of headache and had completely recovered from the paralysis of the left side of the face. The patient stated that his hearing had not improved but that the ability to walk had gradually become better. His disturbance of posture and gait, together with vertigo on quick movements of the head to the face up or the face down position and the jumbling of visual objects were moderate in degree and identical in type with symptoms of patients who suffered of the toxicity of streptomycin therapy.

On vestibular testing, there was no reaction to the rotatory test, even when he was rotated swiftly. There was no reaction to the caloric or the galvanic test in the left ear, and the right ear showed almost a total loss of response. There was total loss of hearing in the left ear, with hearing in the right ear by air conduction for tones of 128 double vibrations at 10 decibels; 256 double vibrations at 20 decibels; 512 double vibrations at 25 decibels; 1024 double vibrations at 30 decibels, and 2048 double vibrations at 60 decibels, above which no tone was heard by air or bone conduction. Air conduction was better than bone conduction.

Roentgenogram of the skull showed only simple fracture of the ridge of the petrous process and the pterygoid process on the left side. Neurologic examination did not disclose any abnormality other than bilateral injury to the eighth cranial nerve. This patient was not seen later than four months after the date of his injury when the regression of symptoms which had taken place was greater than that commonly observed in patients with bilateral vestibular paralysis occurring in streptomycin therapy over a similar duration.

PHYSIOLOGY

Although an explanation of the syndrome of bilateral vestibular paralysis that would bear close scrutiny may be lacking, to no one is there denied at least a glimpse into the problem posed by this disturbance of equilibrium. Because of the ease with which one keeps the proper end of the body uppermost and with which one moves from place to place in such an effortless manner, a disturbance of this function usually bewilders the sufferer and often perplexes the diagnostician. Equilibrial function is perhaps one of the familiar things in life with which man is least familiar.

Physiologists have long recorded that normal posture during rest and movement is attained through effector reflex results of the impulses originating in the vestibular receptors in the inner ear, the visual receptors in the retina of the eye and the exteroceptive and proprioceptive receptors in the skin, muscles, tendons and joints and that the intactness of at least two of these functional systems is necessary to meet the requirements for ordinary activities. Although there is a disturbance of equilibrium following deprivation of the afferent impulses from the eye, skin, muscles, tendons and joints or the vestibular end organ, there is an easily observable qualitative difference in the symptoms, as one may see in the searching gait of the blind for secure footing,

the atoxic gait of the tabetic person, with his obvious dependence on vision, and the staggering gait of persons with disorders of vestibular function. From the loss of any one of these senses there is no severe disturbance of locomotion, but the conclusion from this that no one of these sensory processes has much to do with equilibrium is not valid from the reasoning of Euclid as adapted by Pike,[8] as follows:

> If no one sensory mechanism has much to do with locomotion, it should be possible to eliminate all of them and still retain relatively unimpaired powers of locomotion, for if the whole is equal to the sum of all its parts, the whole effect should be relatively slight. Common experience points very strongly to the conclusion that the whole effect is not relatively slight.

Pike could have added that the effect is more than slight after the loss of any two of these senses.

An unwarranted skepticism has arisen within recent years concerning the usefulness of the vestibular function to man, especially to the aviator. Since the requirement for an applicant for aviation training to comply rigidly to certain standards of reactions in vestibular testing methods in early years of aviation has not proved to be worth while, and as normal vestibular function supports the possibility of vertigo and false sense of direction, the assumption has been made that the ideal aviator would be one with vestibular paralysis.[9]

With the observation that deaf-mutes with vestibular paralysis were immune to vertigo, Hobhouse[10] apparently regretted that the anatomy of the vestibular end organ is such that it cannot be extirpated without causing deafness.

> We are driven to the somewhat painful conclusion that in the semicircular canals man possesses a beautiful and complex mechanism which has been superseded by higher development, and whose only positive function now is to produce some of the most disabling and distressing symptoms which the human body can experience; moreover, this mechanism is so bound up with the organ of hearing that it is impossible to remove it without inflicting penalty of deafness.

On the other hand, many investigators of the functional organization of the vestibular nervous systems have asserted its usefulness in the spontaneous, natural activity of man. Mowrer,[9] in a study of ocular movements in fixation in man, stressed the usefulness of the vestibulo-ocular reflex. In an evaluation of the vestibular function Pike[8] concluded that the resulting disadvantages after ablation of the vestibular receptors (citing the disturbance of equilibrium and the not clearly defined psychologic effects which may be permanent) were more con-

8. Pike, F. H.: Function of the Vestibular Apparatus, Physiol. Rev. **3**:209 (April) 1923.

9. Mowrer, O. H.: Concerning the Normal Function of the Vestibular Apparatus, Ann. Otol., Rhin. & Laryng. **41**:412 (June) 1932.

10. Hobhouse, E.: Aural Vertigo, Lancet **1**:821 (April) 1924.

sequential than the disability caused by infrequent excessive stimulation of the intact vestibular end organ. Moreover, the evidence of symptoms caused by operative lesions in the rhesus monkey is suggestive that after bilateral loss of the vestibular receptors man would still be susceptible to having vertigo and nystagmus from disease involving the vestibular nuclei.[11] The disability of patients with bilateral vestibular paralysis from streptomycin or other causes would seem to be convincing that the vestibular function is not advantageously dispensable in man.

Observations on experimental animals with destructive lesions of the vestibular nervous system have added much to information of clinical sources, even though the analogy to man may not be precise. Beginning with Flourens'[12] discovery of the vestibular receptor of the vestibular reflex nervous system, which has been characterized as the most complex system open to investigators, this system has been studied by many research students. In experimental animals, after bilateral ablation of peripheral vestibular receptors, production of bilateral lesions of the vestibular nuclear complex in the medulla oblongata or extirpation of the entire cerebellum, the disturbances of equilibrium are reported as fundamentally similar in character and resemble closely those of bilateral vestibular paralysis from streptomycin therapy or other causes.

This syndrome was described by Flourens[12] as basically that of the incoordination seen in the gait of a drunken man. Although it may be presumptuous to say that a monkey can suffer from vertigo, nevertheless, in experimental lesions in the rhesus monkey in which both vestibular end organs were destroyed it was noted that in the acute stage the animal, in attempting to jump from the floor to a cage 3 feet (0.9 cm.) above, would turn a back flip and land on its back.[13] This, at least, is suggestive that the animal, on raising its head in the act of jumping, experienced a vertigo (pulsion backward) similar to that of a patient with bilateral vestibular paralysis due to streptomycin. Moreover, such a monkey soon refused to repeat his futile jumping effort and remained inactive, with as much as possible of the surface of his body in contact with the floor of his cage, as if to compensate with additional cutaneous impulses the proprioceptive nervous system for its loss from damage to the vestibular function. This phase of the experimental observation also appears to be in keeping with the comfort that patients with severe

11. Ferraro, A., and Barrera, S. E.: The Effects of Lesions of Various Portions of the Vestibular Complex in the Macaccus Rhesus, Tr. Am. Neurol. A. **64**:69, 1938.

12. Flourens, cited by Fulton and Dow.[14]

13. (*a*) Northington, P., and Barrera, S. E.: Effects of Unilateral and Bilateral Labyrinthectomy and Intracranial Section of Eighth Nerve: Experiments on Monkeys, Arch. Neurol. & Psychiat. **32**:51 (July) 1934. (*b*) Ferraro and Barrera.[11]

vestibular disorder due to streptomycin derive from resting motionless in bed. I also observed that cats after bilateral ablation of vestibular receptors by an operation and cats with vestibular paralysis due to streptomycin showed similar loss of righting reactions on their being dropped with the back downward and, also, that the disorder of gait was similar. Improvement in locomotion was relatively earlier after the traumatic lesion of the ears than after the disability due to streptomycin.

A great similarity of the disturbance of locomotion between bilateral ablation of the vestibular receptors and lesions made in the vestibular parts of the cerebellum in the rhesus monkey are recorded by Fulton and Dow.[14] As described by them, the disequilibrium following removal of the flocculonodular lobe of the cerebellum was brought on by the movement of the body through space, and when the animal was at rest individual movements of the extremities were performed without incoordination or tremor. This syndrome was stated by Dow[14b] to be primarily vestibular because when this part of the cerebellum was destroyed after bilateral deprivation of the vestibular receptors the procedure was symptomless.

The conclusion of de Kleijn and Magnus[15] is informative: "All the labyrinthine reflexes examined (that is to say all the labyrinthine reflexes at present known, with the exception of the reactions to progressive movements) persist unchanged after extirpation of the cerebellum." That is in accord with the disturbance of progressive movement found by Fulton and Dow.[14] The relatively large area of the vestibular nuclear complex in the floor of the fourth ventricle, together with the vestibular connections in the brain stem, is accessible for operative lesions on experimental animals.

As reported from his investigations on the vestibulo-ocular reflex, Lorente de No[16] expressed the belief that the whole vestibular system was a physiologic unit, without localization of reflexes in anatomic nuclei, and that the reflex arc may be closed through one of many pathways but that lesions in any part of the vestibular system modify all reflexes. In support of this explanation, fibers conducting afferent impulses were described as being generally distributed to the vestibular

14. (*a*) Fulton, J. F., and Dow, R. S.: The Cerebellum: A Summary of Functional Localization, Yale J. Biol. & Med. **10**:89 (Oct.) 1937. (*b*) Dow, R. S.: Effect of Lesions in the Vestibular Part of the Cerebellum in Primates, Arch. Neurol. & Psychiat. **40**:500 (Sept.) 1938.

15. de Kleijn, A., and Magnus, R., cited by Camis, M.: Physiology of the Vestibular Apparatus, translated and annotated by R. S. Creed, London, Oxford University Press, 1930.

16. Lorente de No, R.: Vestibulo-Ocular Reflex, Arch. Neurol. & Psychiat. **30**:245 (Aug.) 1933.

nuclear complex, thereby making focal centers within the nuclei improbable. A group of phenomena of the same basic type as those seen in bilateral ablation of vestibular receptors were found by Ferraro and Barrera [11] and by Spiegel [17] in a variety of operative lesions made in the vestibular nuclei in the brain stem. The symptoms caused by the nuclear damage occurred whether or not the vestibular end organs were intact.

The foregoing observations on experimental animals provide evidence that the same basic type of disorder of posture and gait may result from damage of either the vestibular end organs or nuclei in the brain stem or in the cerebellum. There is, however, no claim of which I am aware that injury limited to the cerebellum impairs the vestibular ocular reflex in rotational or caloric testing or impairs the hearing.

The rare type of visual disorder, apparently due to inadequate fixation of objects on movements of the head in bilateral vestibular paralysis in its acute stage, is not in accord with the following physiologic considerations on ocular fixation. Holmes [18] concluded that "in higher animals retinal impulses suffice for normal control of the movements and postures of the eyes when labyrinths or fibers conducting impulses from them are destroyed." Perlman and Case [19] stated that "movement of the eyes may be voluntary (frontal cortex), (2) reflex optic (occipital cortex activated by a retinal image) or (3) reflex vestibular (activated by end organs in the labyrinth)." And it was added that for normal ocular movements the vestibular apparatus is not important.

PATHOLOGY

Histologic examination of serial sections of the temporal bones of the only patient observed by me who had acquired a great loss of hearing along with the usual symptoms of bilateral vestibular paralysis while receiving streptomycin did not reveal pathologic changes in either the cochlear or the vestibular end organs.[20] It has been presumed, however, that the lesion due to streptomycin may be degenerative changes in the vestibular and auditory end organs, since investigations [21] did not disclose pathologic changes in the cerebrum,

17. Spiegel, E. A., and Sommer, I.: Neurology of the Eye, Ear, Nose and Throat, New York, Grune & Stratton, Inc., 1944.

18. Holmes, G.: The Cerebral Integration of the Ocular Movements, Brit. M. J. **2**:4045 (July 16) 1938.

19. Perlman, H. B., and Case, T. J.: Mechanisms of Ocular Movement in Man, Arch. Otolaryng. **40**:457 (Sept.) 1944.

20. Fowler, E. P., Jr.: Personal communication to the author.

21. Muschett, C. W., and Martland, H. S.: Pathologic Changes Resulting from the Administration of Streptomycin, Arch. Path. **42**:619 (Dec.) 1947.

cerebellum or medulla or in the acoustic, brachial or sciatic nerves with use of hematoxylin and eosin, Bodian and Pal-Weigert stains.

It was concluded in one report [22] that streptomycin primarily affects the sensory epithelium of the labyrinth. In this investigation young cats were used and sections were stained with hematoxylin and eosin, Bodian's silver impregnation and Benda-Spielmeyer's myelin sheath stain.

The section shows marked degenerative changes of the sensory epithelium which, normally a columnar epithelium, is now flattened and composed of mere remnants of highly shrunken, degenerated, rounded sensory cells. The nuclei of the supporting cells are displaced basally and form a continuous narrow brim close to the basement membrane. Another remarkable feature is the total absence of cilia. The same marked changes are also seen in the other cristae. . . . Pathological changes similar to those in the cristae ampulares are also found in the sensory epithelium of the macula utriculi and macula sacculi. . . .[22]

Although those pathologic changes were found in all of the 8 animals that received streptomycin and in which the labyrinth was examined, in only 1 were there considerable changes in the organ of Corti.

The histological examination of the brain and especially the vestibular nuclei did not disclose definite pathological changes distinguishable with the usual staining methods.

In the vestibular ganglion of some of the streptomycin animals the Nissl substance of the ganglion cells had a dustlike appearance. These changes, however, were slight and in no way comparable to the marked changes in the sensory epithelium of the labyrinth.[22]

In another report [23] the studies were made on autopsy material, stained by the Nissl's method, from 5 patients who had become partially or totally deaf while receiving streptomycin, and on material from dogs given streptomycin for experimental purposes.

Pathological changes were present in the neurones of the ventral cochlear nuclei in all 5 clinical cases and in all 3 experimental animals, although in 2 of the patients and in one of the animals, these changes were noted in only a few cells. No lesions were noted in the dorsal cochlear nuclei. The vestibular nuclei generally showed no striking change, except for the inferior vestibular nucleus, which in 2 cases showed a similar marked liquefaction necrosis and dropping out of cells and hydropic degeneration. The superior vestibular nuclei were not definitely identified. Other cranial nerve nuclei were examined in every case, often in the same histological preparations as those containing the eighth nerve nuclei. None showed degenerative changes similar to those described. The eighth cranial nerves in the 2 cases studied were normal.

Considered as a whole, the findings suggest that streptomycin can cause a specific destructive effect on the neurones of the eighth cranial nerve nuclei, especially the ventral cochlear nuclei (upon which discriminative hearing depends) and

22. Berg, K.: The Toxic Effect of Streptomycin on the Eighth Cranial Nerve, Ann. Otol., Rhin. & Laryng. **55**:448 (June) 1949.

possibly the inferior vestibular nuclei. These changes would seem to be sufficient in some of the clinical cases to account for deafness and perhaps for vestibular dysfunction.[23]

No mention was made in this report of any disturbance of equilibrium in the patients. In patients with tuberculosis of the central nervous system, one must reckon whether or not the loss of hearing is caused by disease or by streptomycin. The fact remains, however, that pathologic changes were reported in the nuclei of both divisions of the acoustic nerve and, oddly enough, with consistently less changes in the vestibular than in the cochlear nucleus. Although confirmation of those observations in man has not come to my attention, pathologic changes are reported[24] in the brain stem and cerebellum in some of the cats which had received toxic doses of streptomycin. This investigation of the site of the pathologic focus of the toxicity of streptomycin in cats was made by three different methods: by histologic studies of the brain stem and cerebellum after use of vital staining with trypan blue; by routine histologic studies of the brain sections stained by phosphotungstic acid and cresyl violet, and by surgical lesions made in the vestibular nuclei. Winston and his colleagues made the following statements:

Because the prolonged administration of streptomycin frequently causes a disturbance of the vestibular mechanism in man and in certain laboratory animals, we have attempted to locate the site of the injury to the vestibular apparatus by three totally different methods of investigation, using the cat as the experimental animal. Evidence of damage to the central portion of the vestibular apparatus was observed in some of the animals by each method of investigation.

By the vital staining technique, the trypan blue dye was visible grossly in the cerebellum and brain stem in all five animals which had received streptomycin. In three of the five specimens, trypan blue granules were observed microscopically in the nuclei of some of the brain cells in the following locations: (1) the Purkinje cells of the cerebellar cortex, most marked in the flocculus; (2) the dentate and fastigial nuclei; (3) the medial and lateral vestibular nuclei. In addition, occasional cells containing nuclear trypan blue granules were found in the ventral cochlear nuclei, the tuberculum acusticum, the nuclei of the trapezoid body, and the recticular substance. In the three control cats, there was neither macroscopic nor microscopic staining of the brain tissue, although the meninges and the choroid plexus were stained a deep blue throughout.

Following unilateral surgical destruction of the vestibular nuclei, the control animal showed the proper nystagmus response to the opposite side. In two cats which had received toxic doses of streptomycin, destruction of the nuclei on one

23. Stevenson, D.; Alvord, C., and Correll, W.: Degeneration and Necrosis of Neurones in Eighth Cranial Nuclei Caused by Streptomycin, Proc. Soc. Exper. Biol. & Med. **65**:86 (May) 1947.

24. Winston, J., and others: An Experimental Study of the Toxic Effects of Streptomycin on the Vestibular Apparatus of the Cat: Central Nervous System, Ann. Otol., Rhin. & Laryng. **57**:738 (Sept.) 1948.

side failed to produce nystagmus. This was interpreted as evidence that streptomycin destroys the function of the vestibular nuclei on both sides of the brain stem. However, these findings do not rule out the possibility that the peripheral end-organs of the vestibular apparatus may be damaged also.

Routine histopathologic studies of brain sections stained with phosphotungstic acid and cresyl violet were done on six additional cats which had been given large doses of streptomycin. Pyknosis and an increase of glial fibers around the dendrites of the Purkinje cells of the cerebellum were found in four of the six brains. In the remaining two specimens, no pathologic changes were found.[24]

It was added that the authors' experimental findings did not exclude the possibility of concomitant damage to the vestibular and organs.

By quantitative cytochemical methods the distribution and content of nucleic acids and proteins of the vestibular ganglion and Deiters' nucleus in guinea pigs after they had received toxic doses of streptomycin were reported.[25]

> Streptomycin . . . causes disturbances in the intracellular chemical processes in the nerve cells. These disturbances are extensive and can easily be established by means of quantitative cytochemical methods. They consist of a decrease of the nucleoprotein content in the cytoplasms of the nerve cells, showing an inhibition of the intracellular production of these substances. This phenomenon can be correlated to the cessation of vestibular function. . . .
>
> Conclusions can also be drawn from our experiments regarding the site of attack of the streptomycin, i. e. in the periphery or the centre of the vestibular pathway. The most extensive changes take place in the vestibular ganglion. In one of the second neurons, i. e. in Deiters' nucleus, the changes are evident and far more serious than those found in connexion with fatigue of the vestibular apparatus. . . .
>
> The effect of streptomycin is interpreted as an inhibition of the intracellular reproduction of nucleic acids.
>
> This effect appears to be electively bound to the nerve cells belonging to the vestibular pathway.[25]

The following account seems to leave no doubt of the possibility of disseminated lesions of the central nervous system being caused by streptomycin. The only recorded death[26] found in a review of the literature which was attributed to streptomycin was that of a patient who had a concentration of 250 micrograms in each cubic centimeter of blood serum after a total of only 15 Gm. (225 grains) of the drug had been administered over five days, 3.2 Gm. (50 grains) being given daily in six doses, when alarming symptoms occurred with dramatic

25. Floberg, L. E.; Hamberger, C. A., and Hydén, H.: Inhibition of Nucleic Acid Production in Vestibular Nerve Cells by Streptomycin, Acta oto-laryng., 1949, supp. 75, p. 36.

26. Hunnicutt, T.; Graf, W. J.; Hamburger, M.; Ferris, E. B., and Scheinker, I. M.: Fatal Toxic Encephalopathy Apparently Caused by Streptomycin, J. A. M. A. **137**:599 (June 12) 1948.

suddenness: Disorientation and choreiform and athetoid movements were followed by generalized convulsions, hyperpyrexia, tachycardia, arterial hypertension and Cheyne-Stokes respiration. Death occurred on the eighth day after the beginning of streptomycin therapy. The neuropathologic changes were described as

> . . . functional vascular disturbances of the white matter; medulla and basal ganglions characteristic of vasoparalysis, associated with secondary cerebral swelling and ischemic alterations of the nerve cells of the nuclei and of the cortical gray matter; petechial hemorrhages in the paraventricular and supraoptic nuclei of the hypothalamus.[26]

This summary of a comprehensive neurohistologic examination discloses the extent of the area of the brain which may be vulnerable to streptomycin; yet none of the patients whom I have seen exhibited symptoms, except for transitory paresthesias about the face, which indicated impairment of function of neurologic elements other than that of the eighth cranial nerve.

COMMENT

In evaluating the reactions in aural testing in patients with neurotoxic symptoms due to streptomycin in the light of previous experiences in vestibular disorders, the findings were not consistent with those commonly present when the lesion was either exclusively in the peripheral segments or exclusively in the central segments of the vestibular nervous system. The reactions on aural functional tests, therefore, are in keeping with the assumption that may be made from the pathologic reports cited that streptomycin may damage both peripheral and central components of both divisions of the acoustic nerve.

Although the disorder of equilibrium and of vision caused by streptomycin and by bilateral ablation of the vestibular end organs is of the same type as that following peripheral lesions, there was total loss of the vestibulo-ocular reflex on rotatory and caloric testing, and recovery from the severe disability in the acute stage was earlier both in man and in experimental animals than in the disturbance of equilibrium from streptomycin. In addition, in no patient was there a permanent total loss of the vestibulo-ocular reflex; nor was there always a parallel between the degree of disequilibrium and the impairment of the vestibulo-ocular reflex, as shown on functional aural tests.

Streptomycin so rarely caused deafness along with impairment of vestibular reactions that belief in a central lesion is prompted because of the common experience that the cochlear functional loss is regularly seen with peripheral vestibular disorders. Another usual finding which was not consistent with peripheral vestibular disease was the difference in degree of reactions in vestibular testing methods when no reaction

was obtained in some patients after rotation, even at a high speed, and responses of nystagmus, vertigo and past pointing in caloric testing were relatively good.

The most frequent change was an early impairment, increasing to total loss, of reactions to the rotatory test in the acute stage soon after the appearance of disturbed equilibrium, accompanied with only a gradual loss on caloric testing, which in some subjects reached a total loss after the acute stage. In the chronic stage, with improvement of vestibular reactions, reactions were usually more active in caloric tests than in rotatory tests. This lack of similarity in degree of reactions to rotatory and caloric testing from a lesion of the peripheral portions of the vestibular nerve does not conform to clinical experience.

It is, of course, to be kept in mind that in this association unilateral ablation of the function of one vestibular receptor will be followed in the chronic stage by an equal nystagmic response on right and left rotatory testing, but one of shorter duration than when both vestibular receptors were intact. On the other hand, when reactions in vestibular testing were obtained the responses of nystagmus, vertigo and past pointing were normal in type. In no patient was there observed a spontaneous nystagmus or a nystagmus induced showing qualitative changes, i. e., perverted nystagmus, dissociated ocular movements or ocular deviations, symptoms commonly present in disease of the central component of the vestibular nervous system.

It is of interest to cite a case of encephalitis with bilateral vestibular paralysis reported by Spiller and Jones.[27] The patient in question had a spontaneous nystagmus of indefinite character, with eyes constantly in motion through a small range and a vertical nystagmus on looking upward. The spontaneous phenomena were not changed in caloric or rotatory testing. The patient's cochlear function was normal. Histologic examination was made of serial sections of the brain stem from the lower portion of the medulla oblongata to the end of the aqueduct. Damage found in this case extended beyond the vestibular nuclei to connections with the posterior longitudinal bundles. Abnormal histologic states as the effect of streptomycin have not been reported in this region, and this lack of vulnerability may account for the ocular symptoms varying in the patient cited from those of patients with bilateral vestibular paralysis caused by streptomycin.

The optokinetic nystagmus was noted in all of the several patients tested, without any variation in the reaction of those who had or had not any reactions in the vestibular tests, although the efferent impulses

27. Jones, I. H., and Spiller, W. G.: The Central Tracts of the Nervus Vestibularis, Brain **48**:334 (Sept.) 1925.

in this reflex action have been reported as mediated by the vestibular nuclei,[17] an area apparently vulnerable to the neurotoxic effect of streptomycin.

The subjective symptoms, jumbling of visual objects and vertigo, and their influence on the chief disability disturbance of locomotion are perplexing. They regularly occurred in bilateral vestibular paralysis of known peripheral cause from operative lesions as well as from streptomycin. Mowrer[9] implied that precision of ocular movements whereby fixation on shifting the gaze with rotation of the head, alternately to right and to left to view objects, one set at 45 degrees to his left and the other at 45 degrees to his right, is made possible by vestibulo-ocular reflex action.

Ford and Walsh[28] reported detectable variations from normal ocular movements in certain movements of the head in patients with bilateral vestibular paralysis. I have been unable to detect abnormal movements of the eyes in the patients who complained of jumbling of visual objects, but investigation of this symptom is not ended.

Although cochlear function may be impaired by streptomycin, only rarely did patients have any loss of hearing. Information on deafness of central origin is largely hypothetical,[29] but a case heretofore was cited in which almost total loss of hearing with comparable vestibular loss occurred, and later histologic examination of serial sections of the temporal bones did not reveal abnormal changes in the cochlear or vestibular elements. It was mentioned previously that in one report histologic changes were commoner in the cochlear nuclei than in the vestibular after administration of streptomycin. These findings prompt the interesting speculation that deafness often attributed to uncertain aural diseases may in some instances be caused by pathologic changes in the central segments of the cochlear nervous system.

SUMMARY AND CONCLUSIONS

Fifty-six patients were observed who experienced symptoms which were suggestive of bilateral vestibular paralysis from toxicity of streptomycin. Six had other pathologic conditions to account for their disabilities. In only 1 did streptomycin cause impairment of hearing sufficient for it to be detected by the patient. All patients receiving a daily dosage of 2.5 Gm. (37.5 grains) to 3.2 Gm. (50 grains) of streptomycin had neurotoxic effects of the drug, which usually occurred during the second week of its administration. Fifty patients were

28. Ford, F. R., and Walsh, F. B.: Clinical Observations upon the Importance of the Vestibular Reflexes in Ocular Movements: The Effects of Section of One or Both Vestibular Nerves, Bull. Johns Hopkins Hosp. **58**:80 (Feb.) 1936.

29. Northington, P.: Hearing in Patients with Intracranial Tumors, Bull. Neurol. Inst. New York **5**:289 (Aug.) 1936.

observed who received a daily dosage of 1.0 Gm. (15 grains) of streptomycin for seventy-five consecutive days with only 1 experiencing neurotoxic effects.

The symptoms of bilateral vestibular paralysis conform to a pattern, mainly, a swaying posture, staggering gait and jumbling of visual objects on head movements, with or without body movements, and impairment or total loss of reactions on vestibular testing, with or without a hearing loss. This syndrome may be caused by damage to either the peripheral or the central segments of the vestibular nervous system.[2] The acute symptoms tend to subside earlier and the degree of compensation is greater in the chronic stage when the cause is known to be peripherally located, as from traumatic destruction of the vestibular receptors, than when due to streptomycin.

Although the reports cited of physiologic and pathologic investigations indicate that the basic type of disequilibrium shown in bilateral vestibular paralysis may result from focal lesions of the vestibular nervous system in either its peripheral segments or its central nuclear segments in the medulla oblongata or the cerebellum, it is not in accord with acceptable information that a disorder of function of the cerebellum modifies either the cochlear function or the vestibulo-ocular reflex. The impairment of the vestibulo-ocular reflex occurring regularly and the cochlear reflex occasionally in patients with neurotoxic symptoms due to streptomycin permits the exclusion of the cerebellum as its chief focal lesion. The visual disorder regularly present in patients with bilateral vestibular paralysis is evidence that intactness of the vestibulo-ocular reflex is of greater importance than generally assumed in fixation of visual objects.

Although it is customary to observe a good degree of compensation after total and permanent loss of the aid of afferent impulses of any one of the sensory nervous systems contributing to equilibratory function, there may be a severe disturbance of equilibrium, due to streptomycin without a total loss of even one sensory function—the vestibular. A good degree of compensation has been noted, moreover, to occur early in some patients in the presence of gross loss of reactions on vestibular tests and slowly in others when such reactions were relatively good. It is concluded, therefore, that the coordinating and integrating processes of afferent impulses into terms of muscular activities are damaged by streptomycin and that intactness of the central vestibular components is of greater importance to maintenance of equilibrium than normal afferent sensory systems.

The outlook is promising for the selective neurotoxic action of streptomycin to provide the basic needs for investigations in experimental animals, to the extent not heretofore possible, of perplexing problems of the vestibular and cochlear reflexes.

A STUDY OF THE DEAFNESS HERITAGE IN OTOSCLEROSIS

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IN AN EARLIER contribution to the study of deafness in 581 cases, in which 461 biochemical examinations of the blood were undertaken, there were a large number of cases with the criteria to warrant a diagnosis of otosclerosis.[1] Among these otosclerotic patients were groups of deafened sisters and deafened mothers with deafened children.

In the familial groups, the biochemical findings in the blood were so significantly similar, irrespective of the differences in age of the blood relatives, that a specific study of the heritage of deafness in otosclerosis was begun, particularly so because of the pertinent biochemical abnormality in the blood, which was demonstrated by the first study of the blood of both parents and of the deafened child, of which I have knowledge. These parents, although totally unconscious of having any loss in hearing acuity, nevertheless showed in the pathologic picture of their blood that they carried a metabolic mechanism defect, which they transmitted to their child, who was a congenital deaf-mute.

In table 1 are shown the pertinent blood findings of this group.

The child exhibited both hyperpyruvemia and hypercholesteremia. The father exhibited only hyperpyruvemia, and the mother's blood showed both hyperpyruvemia and hypercholesteremia, as did the child's.

In table 2 are shown, in juxtaposition, the blood biochemical findings of three sets of sisters and of two unrelated mothers and their deafened children.

The sisters, A. M. and M. C., showed only hyperpyruvemia. All the other patients mentioned in table 2, the sisters and the mothers and their respective children, exhibited hyperpyruvemia and hypercholesteremia. Their presence signifies a carbohydrate metabolic defect in the phos-

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1. Kopetzky, S. J.: The Reversal of Biochemical Processes in Cases of Cochlear and Vestibular Dysfunction, J. Internat. Coll. Surgeons **13**:139 (Feb.) 1950.

phorylation mechanism and, in addition to this, a lack of lipotropic factors in liver function, in those cases particularly in which both hyperpyruvemia and hypercholesteremia are exhibited.

I conclude from this earlier study of 581 cases of deafness [1] that this chemical abnormality in the blood actually comprises the factors at fault in the evolution of the lesions of otosclerosis.

In contrast to the factors at fault [2] found in cases of deafness due to lesions of otosclerosis—especially since these factors are found in

TABLE 1.—*Blood Findings for Deaf-Mute Aged 3 and for Both Parents*

Item	Normal	Child's Blood	Mother's Blood	Father's Blood
B.P. Pyruvic acid	0.6	1.55	0.8	0.7
B.S. Cholesterol	160-200 *	280	207.5 †	196.0 ‡

* Normal for child is 160 to 190 mg. per hundred cubic centimeters; for adult, 160 to 200.
† Mother's hearing is at low normal (10 decibel level).
‡ Father is deafened when audiometrically tested but is not conscious of hearing loss.
(B.P. indicates blood plasma. B.S. indicates blood serum.)

TABLE 2.—*Blood Findings of Three Sets of Sisters and of Two Unrelated Mothers and Their Deafened Children*

Blood Relationship	Case	Age, Yr.	Blood Plasma Pyruvic Acid	Blood Serum Cholesterol
Sisters	A. M.	29	0.816	180
	M. C.	23	0.820	177
Sisters	S. L.	27	0.816	215
	B. L.	33	0.786	452
Sisters	I. B.	45	0.826	215
	A. L.	37	0.906	280
Mother	C. T.	40	0.6	280
Daughter	S. T.	9	1.78	305.0 *
Mother	L. R.	33	0.786	280
Son	D. R.	6	0.786	260 *

* Normal for child is 160 to 190 mg. per hundred cubic centimeters; for adult, 160 to 200.

the blood of not only the greatly deafened children and deaf-mutes but also in the blood of their respective parents, who are not conscious of being deafened yet who carry in their blood hyperpyruvemia alone or hyperpyruvemia and hypercholesteremia—I present two illustrative cases of deafness which are not of otosclerotic origin, with the blood biochemistry findings.

One patient, D. W., aged 53 years, suffers from a large defect in the membrana tympani due to cholesteatoma in one ear, which has been dry for many years, and,

2. Kopetzky, S. J.: Deafness, Tinnitus and Vertigo, New York, Thos. Nelson & Sons, 1948, chap. 7 and chap. 10, pt. 9.

in the other, from the sequelae of a middle ear infection with otoscopically evident adhesive bands. The audiometric chart of air conduction is shown in figure 1.

The audiometric tests for bone conduction under conditions of masking reveal an acoustic nerve type of hearing dysfunction.

The blood biochemistry findings were normal for pyruvic acid, 0.6 mg. per hundred cubic centimeters, and the total cholesterol was 185.0 mg. per hundred cubic centimeters, which is also in the normal range.

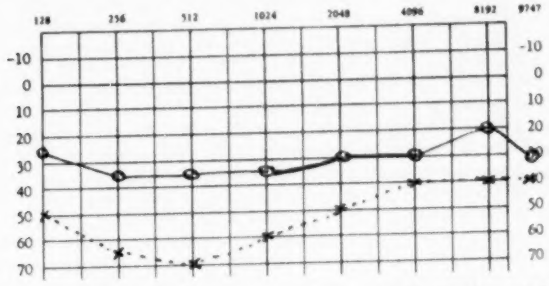

Fig. 1.—Audiometric chart for patient D. W., aged 53, showing whole range hearing loss. The unbroken line with circles indicates a loss of 26⅝ per cent for the right ear; the broken line with crosses indicates a loss of 46⅞ per cent for left ear.

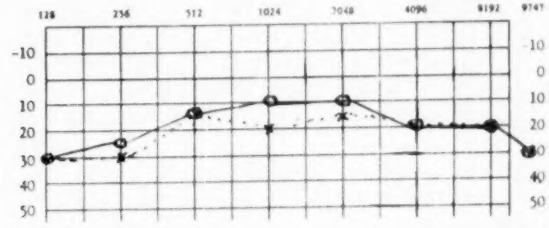

Fig. 2.—Audiometric chart for patient L. G., aged 46, showing whole range hearing loss. The unbroken line with circle indicates a loss of 18¾ per cent for the right ear; the broken line with crosses indicates a loss of 19⅜ per cent for the left ear.

The second case is that of a woman, L. G., 46 years of age, whose chief complaint is a hearing loss (see figure 2), with a history of abscessed ears twenty-seven years ago, the ears since then having been dry. The most pertinent finding was a histamine hypersensitivity. Biochemical examinations of the blood indicated no abnormalities. The pyruvic acid was 0.6 mg. per hundred cubic centimeters, which is normal, and the total cholesterol was 200 mg. per hundred cubic centimeters, which is also normal according to my laboratory.

These charts, particularly the second one, are very significant when one compares the slight hearing loss shown in L. G.'s case with the hearing results of the deafened children's parents, who are not conscious of their slight hearing losses

yet who show the typical otosclerotic abnormality in the blood, which their greatly deafened and deaf-mute children also exhibit[2]; but these findings are absent in the sample control cases, those with deafness of nonotosclerotic origin.

Mr. Nicholas Molnar, chemist, who supervised my chemical work, gives the following additional instructions: In the test methods for pyruvic acid, the reading must be taken after the last reduction process, at the end of ten minutes, no later or no earlier, otherwise false values will result. In addition, the patient must be completely relaxed and rested before the blood specimen is withdrawn.

With regard to testing for blood cholesterol, some very fine points in chemical technic are necessary. Great care is to be taken that the blood serum introduced into the extraction mixture is introduced slowly, by rocking both the flask and the pipet in order to break up any lumps that may form. When the serum is introduced in very small drops and the solvent vigorously agitated in the flask, the protein precipitated appears in the form of a powder. Thereby the extraction of the cholesterol and total lipids by the solvent is far easier and is complete. If the serum is introduced fast, or if there is no agitation of the flask, a lump is formed as the protein-rich serum hits the extraction mixture, which consists of alcohol and ether. Then the extraction is not proper and is incomplete, and the results will be variable. There is one other precaution: The solvent should be heated to its boiling point and allowed to cool to room temperature, then brought to proper volume. The extraction must take place at room or incubator temperature, otherwise complete extraction will not be obtained; the results will be variable. In my laboratory the final filtering process, with a stoppered centrifuge tube, is used to prevent evaporation and loss of solvent; otherwise the results are variable.

A pathologic change of blood chemistry precedes the evolution of pathologic cellular changes in otosclerosis. The blood findings help to explain the clinical test results which show a type of deafness in which the perception of sound wave stimuli is hindered, as presented in the first phase of otosclerotic evolution. The energy of a sound wave pattern is not adequately transformed at this time into a nervous impulse, received at the end organ of the acoustic nerve and transmitted centrally along the nerve synapses. This happens long before a conduction hindrance to sound wave transmission occurs, because pathologic change of the cells has not as yet involved the labyrinthine windows, to lessen their elasticity, nor as yet formed abnormal calcium deposits in these membranes, to fix either one or both of the labyrinthine windows and clinically exhibit an impeded or an immobile ossicular chain. There is one exception to this statement, namely, that in my series of cases, in 1 of every 5

3. The tests for pyruvic acid are those described by Lu, G. D.: Studies on the Metabolism of Pyruvic Acid in Normal and Vitamin B_1-Deficient States: A Rapid, Specific and Sensitive Method for the Estimation of Blood Pyruvate, Biochem. J. **23**:249, 1939; Bueding, E., and Wortis, H.: The Stabilization and Determination of Pyruvic Acid in the Blood, J. Biol. Chem. **133**:585, 1940. Friedemann, T. E., and Haugen, G. E.: Pyruvic Acid: The Determination of Veto Acids in Blood and Urine, ibid. **147**:415, 1943. The tests for determining cholesterol are shown in Bloor, W. R.: The Determination of Cholesterol in Blood, J. Biol. Chem. **24**:227, 1916; Stecher, R. M., and Hersh, A. H.: Notes on the Genetics of Hypercholesterolemia, Science **109**:61, 1949.

histamine hypersensitivity was presented. This hypersensitivity conceivably can, and often does, present indurations of the reduplicated membranous folds of Prussak's space and the ossicular suspensory ligaments as well involve the lining mucosal membrane of the tympanic cavity and of the eustachian tube. The tissues of the nasopharyngeal space are also involved. This involvement creates an impediment to sound wave transmission also.

Otosclerosis is a disease with lesions on the otic capsule. When such lesions develop at sites where by their presence they hinder physiologic function of the hearing apparatus, either because of structural abnormalities or by interfering with the perception of sound wave stimuli, it is a symptomatic disease. Its dominant symptom is a progressive type of deafness, and the patient is conscious of his hearing loss.

There is another form of otosclerosis. It is usually found only at autopsy. During life the patient is not conscious of being deafened. He not only never complains of being deafened but when directly questioned even denies having any difficulty in hearing.

At autopsy, histopathologic examinations of the labyrinthine structures taken from patients who died from some disease not associated with the auditory apparatus revealed the characteristic otosclerotic lesions on the otic capsule. Such lesions however, do not invade or involve the physiologically functioning portions of the hearing apparatus up to the time of the patients' death. Figure 3 is a photomicrograph of a section showing such an otosclerotic lesion on the promontory, located adjacent to but not involving the round window. This specimen was taken from a person with normal hearing and was reported by Brühl [4a] as an accidental finding.

During the course of these studies in otosclerosis there have been occasions when an examination was made on numbers of deafened children and deaf-mutes. The parents of these children apparently had normal hearing. In taking the little patients' history, there frequently were times when a positive familial history was frankly stated. In some cases the mother's blood relatives were said to be deafened; in others, the father's relatives were. In a few cases both sides of the family carried the history of deafness suffered by the grandparents on both sides. In some instances, even the otologist who had treated the grandparents was known to me as a competent otologist whose diagnosis of otosclerosis could be accepted without reservation.

4. (a) Brühl, G.: The Pathology of Asymptomatic Otosclerosis, in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen-, Ohrenheilkunde, mit Einschluss der Grenzgebiete, Berlin, Springer-Verlag, 1929, vol. 7, pt. 2, pp. 410 and 432. (*b*) Manasse, P.: Otosclerosis, Vienna, Alfred Holder, 1917; also reports to the German Otological Society at Basal, Switzerland, 1909 and 1914. (*c*) Grünberg, K.: Zur Pathogenese der Otosklerose, Ztschr. f. Laryngol. u. Rhinol. **15**:78 (Nov.) 1926.

In other instances, a family history of deafness in the blood lines of the child was strongly denied. Notwithstanding these denials and the added information of the parents that they suffered no hearing loss, audiometric hearing tests were undertaken on some of these parents.[5] In most of the examinations made on such parents there were revealed hearing losses for pure tones by audiometric tests, either above or below the frequency cycles which comprise the speech range of hearing.

Thus the unconsciousness of hearing loss becomes comprehensible because the sound waves for speech were adequately heard. However, biochemical examinations of the blood of the parents indicated the presence of hyperpyruvemia either alone or with hypercholesteremia in the father or the mother, or both. Because of this fact, in the absence of other conditions or diseases[6] that might account for the abnormality in

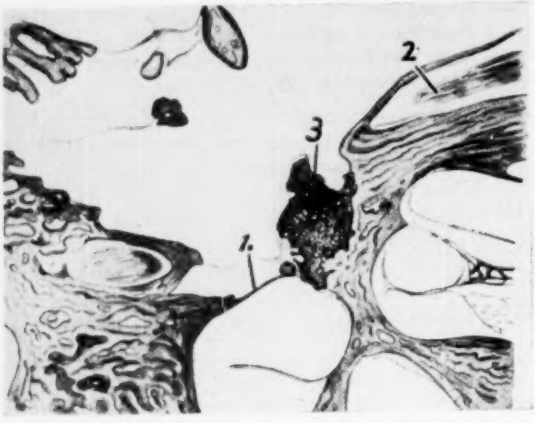

Fig. 3.—Photomicrograph of an otosclerotic lesion on the promontory in a person with normal hearing. *1* indicates the labyrinthian round window; *2*, the canal of the tensor tympani muscle, and *3*, the otosclerotic lesion on the promontory, near the fenestra rotunda. (From Brühl.[4b])

5. In spite of all explanations, some parents of even deaf-mute children refused to cooperate or to submit to personal hearing tests or examinations of the blood, even when they were offered gratis.

6. Before assigning the diagnosis of asymptomatic otosclerosis to parents or relatives of deafened children and deaf-mutes on the basis of the presence of hyperpyruvemia alone or of hyperpyruvemia and hypercholesteremia, the physician must assure himself that no other conditions or diseases exist in these blood relatives not associated with otosclerosis that would carry the hyperpyruvemia or the hypercholesteremia as part of the laboratory findings. Such diseases and conditions are arteriosclerosis, coronary thrombosis, biliary obstruction, diabetes, clinical hypothyroidism, nephrosis, chronic glomerulonephritis, advanced pregnancy, xanthomatosis and cancerous neoplasms, particularly those in the gastrointestinal tract.

the blood, especially in those cases in which a familial history of deafness is obtainable, this biochemical abnormality permits a diagnosis of asymptomatic otosclerosis in the parents.[4] They carry in their blood one or both of the factors at fault which I have found present in the blood in cases of symptomatic otosclerosis.

Such a postulate heretofore has not been possible, scientifically.

In the sample genealogic charts of the blood lines of representative cases (figure 4 through figure 12) are exhibited the biochemical findings in the blood in conjunction with the relationship of the parents to the patient and to the known history of familial deafness.

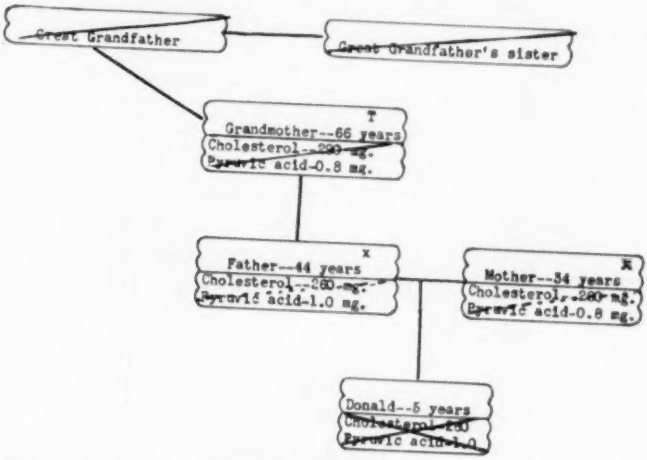

Fig. 4.—Self-explanatory genealogic chart of male child aged 5. The deaf-mute child inherits from both parents, but the heritage is strongest from the father's blood line. Both parents are not conscious of their hearing losses. The broken diagonal line indicates slight hearing loss; the unbroken diagonal line, severe hearing loss, and the crossed diagonal lines, deaf-mutism. *X* indicates asymptomatic otosclerosis, and *T*, symptomatic otosclerosis.

Based on the premises which are postulated here—the importance of biochemical examinations of the blood of other siblings in a family in which one sibling exhibits an inherited deafness, with biochemical findings in the blood of the factors at fault usually presented in otosclerotic deafness—it follows that any intercurrent severe systemic infection or the presence of puberty, pregnancy or menopause may present the locally irritant factor which will start a cellular pathologic condition into activity and create symptomatic otosclerosis in the sisters or brothers of the deaf-mute child.

In addition to enabling me to formulate the preceding postulate regarding the ability to diagnose asymptomatic otosclerosis from the biochemical abnormality of the blood of the living patient, it is now also possible to negate the idea, which many reports have assumed about heredity as an etiologic factor, that the genealogic charts often exhibit a skipped generation in the transmission of the heritage along the direct blood lines of the family. If the biochemical examinations of the blood are performed, with the positive finding of the factors at fault, as out-

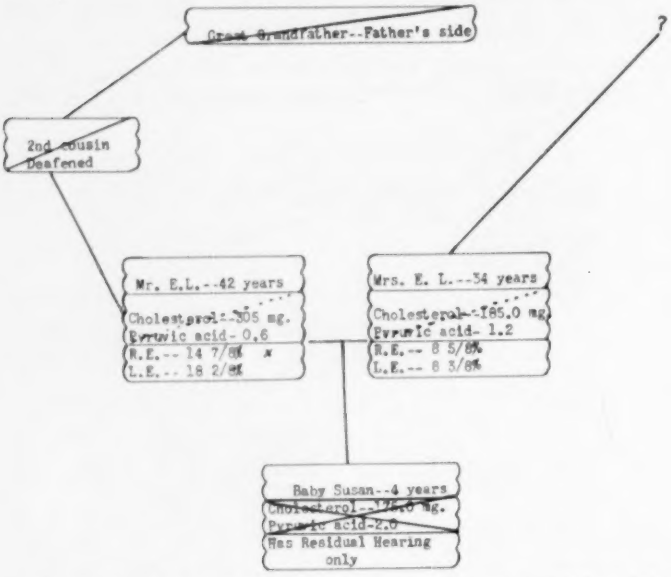

Fig. 5.—Genealogic chart of a deaf-mute female child aged 4. Neither of the parents is conscious of deafness. The blood of both the mother and the child exhibits only hyperpyruvemia, but the father's blood lines carry the major factors of the heritage of deafness, since his blood exhibits both hyperpyruvemia and hypercholesteremia, corresponding to the known family history of deafness. The normal range for cholesterol in the adult is 160 to 200 mg. per hundred cubic centimeters and in the child, 160 to 190. The normal range for pyruvic acid in the adult is 0.6 mg.

The broken diagonal line indicates slight loss of hearing; the unbroken line, considerable hearing loss, and the crossed lines, deaf-mutism. *X* indicates asymptomatic otosclerosis.

lined here, any statement of parents and relatives of the deafened child or deaf-mute may be disregarded. If the deaf-mute child and the blood relatives of the child exhibit in their respective blood specimens the factors at fault in otosclerosis, skipped generations will not be presented.

The so-called skipped generation most probably, in most genealogic charts on record, would, if their blood chemistry had been studied, be found to have asymptomatic otosclerosis.

One more item needs notation: The asymptomatic otosclerosis of today, with its slight hearing losses above and below the speech range frequencies, can, and often does, change to symptomatic otosclerosis when a locally irritant factor comes into the pathologic picture. To discuss this item further would lead too far afield at this time. This fact, however, needs elucidation here to make comprehensible what follows, which concerns the heritage of otosclerosis particularly.

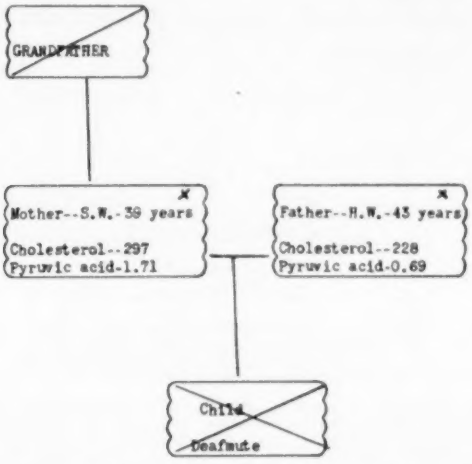

Fig. 6.—Self-explanatory genealogic chart of a congenital deaf-mute child who died of encephalitis one year prior to the time when the parents voluntarily submitted themselves to otoscopic and functional examinations and biochemical examinations of the blood. The child inherited from both parents, neither of whom are conscious of being deafened. The mother's father was deafened. The parents probably have asymptomatic otosclerosis. No tuning fork tests were given to these parents. The father, aged 43, has impeded ossicles in the left ear. The mother, aged 39, had freely movable ossicles. Her grandfather was deafened since childhood. The father's history reveals diabetes in his maternal grandmother. He had had rheumatic fever in childhood. The diagonal line indicates deafened; the crossed lines, deaf-mutism, and the absence of any lines indicates normal hearing.

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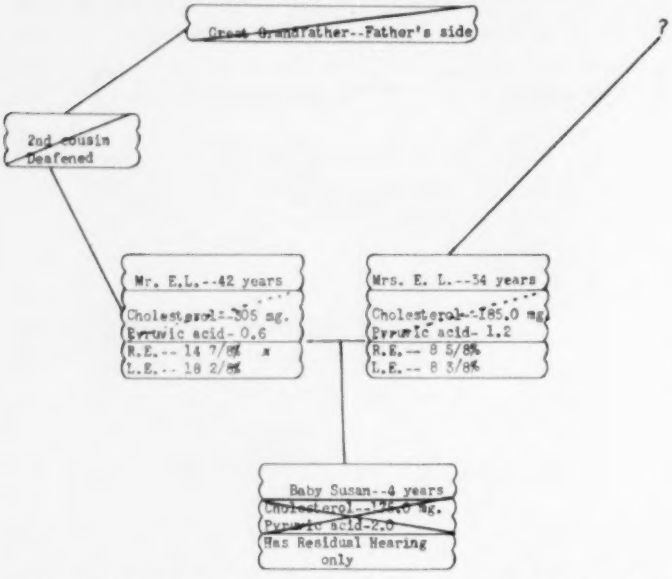

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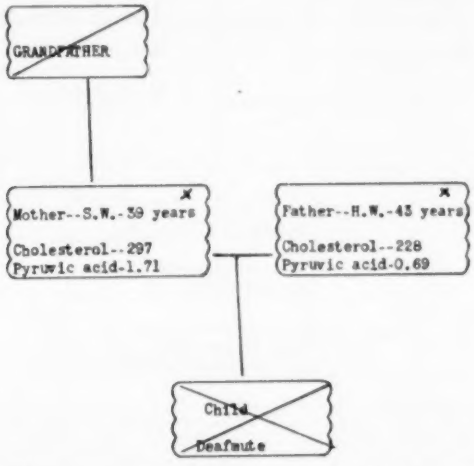

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Finally, from the clinical angle, with a knowledge of what the biochemical abnormalities in the blood signify, it is possible to postulate a theory regarding the mechanics by which hereditary factors starting in intrauterine life determine the growth of bone in general and that of the otic capsule in particular.

The congenitally deafened are those to whom deafness has been transmitted by birth.[7] They are born with an imperfection or with the implantation of a pathologic process. There may be either a biologic dysfunction or a dysfunction which may have started a pathologic lesion in intrauterine life.

Since 1860, genealogic charts have disclosed the heritage of deafness as an etiologic factor in otosclerosis,[8] and identical twins have been

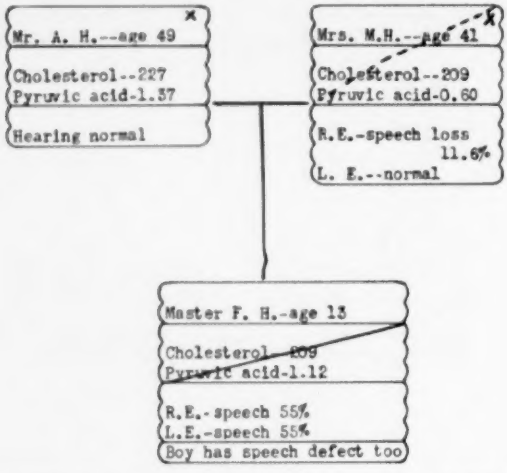

Fig. 7.—Blood line of a youth, aged 13, who has approximately one half of his hearing acuity. His ossicles are movable; his father's are sluggish. His heritage of deafness stems from both parents, although the father's audiometric tests show normal hearing, but the father exhibits hyperpyruvemia and hypercholesteremia and is otherwise physically healthy; the mother exhibits only hyperpyruvemia. The child shows both abnormalities in the blood, according to the laboratory standards used. Both parents are asymptomatic with regard to deafness. His father, A. H., aged 49, had a hypertensive condition. His mother, M. H., aged 41, had had a high forceps delivery with the boy. The broken diagonal line indicates unconsciousness of deafness; the unbroken line indicates evident hearing loss and speech difficulties, (heritage from both sides of the family), and absence of lines indicates normal hearing. *X* indicates asymptomatic otosclerosis.

7. One is not dealing here with congenital deaf-mutism due to traumatic birth injuries or to intracranial hemorrhages affecting the cortical motor zone of the brain, or with congenital deaf-mutism due to congenital syphilis involving the internal ear or due to other intercurrent causation. Here one is specifically concerned with inherited otosclerosis.

8. Duel, A. B.: Heritage: An Etiological Factor in Otosclerosis; A Résumé of the Literature to 1928, New York, Paul P. Hoeber, Inc., 1929, vol. 1, sect. 11, chap. 5.

found to present analogous findings.[9] Recently, a study in labyrinthine deafness and paroxysmal vertigo by Brown[10] was summarized with a statement that these appear to be inherited diseases.

What the heritage is and how it comes into being in succeeding generations of a family has not heretofore been postulated.

The older literature usually contents itself with vague references to "inheriting a tendency toward [deafness]"—*eine Neigung dazu.* The startling finding in the biochemical examination of the blood made on the little deaf-mute girl and her parents (fig. 1) led to the deductions that permitted a comprehension of the elements which "set the stage," as it were, for the onset and development of the cellular pathologic process on the otic capsule that, when evolved, is designated as the pathologic lesions of otosclerosis.

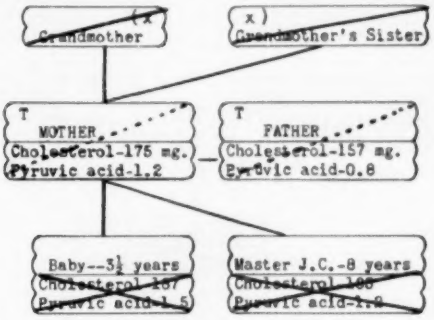

Fig. 8.—Blood lines of 2 deaf-mute children (8 and 3½ years) in one family. Those of the family who were examined exhibited hyperpyruvemia only, the indicator of a defect in the phosphorylation mechanisms of carbohydrate metabolism. The baby, 3½ years, is too young to undergo functional testing; she seems to give some response to sound stimuli but does not talk. The brother, aged 8, is also a mute. Both parents show slight hearing losses on testing but are not conscious of deafness. On the mother's side, her mother and the mother's aunt were deafened and conscious of it. The unbroken diagonal line indicates marked hearing loss; the broken line, slight hearing loss with no consciousness of deafness, and the crossed lines indicate deaf-mutism. *X* indicates diagnosis of otosclerosis; *T* indicates slight deafness on tests but no consciousness of deafness (asymptomatic otosclerosis).

The factors which are transmitted as the heritage of otosclerotic deafness are those affecting the chemical processes in enzymatic and coenzymatic dysfunction, secondary to a defect or deficiency in the

9. Kerrison, P. D.: Diseases of the Ear, Philadelphia, J. B. Lippincott Company, 1913, p. 185. Fowler, E. P.: Otosclerosis in Identical Twins: Five Case Histories, Ann. Otol., Rhin. & Laryng. **58**:365 (June) 1947.

10. Brown, M. R.: The Factor of Heredity in Labyrinthine Deafness and Paroxysmal Vertigo (Ménière's Syndrome), Ann. Otol., Rhin. & Laryng. **58**:665 (Sept.) 1949.

phosphorylation mechanism of carbohydrate metabolism, of which diphosphothiamine-magnesium-protein is one of the principal deficiency items. Because of this, and before the cellular pathologic change has reached the functioning elements concerned with hearing, there is already dysfunction of the perceptive faculty of the acoustic end organ. This situation has been noted by many observers, from Gray [11] to Brown.[10]

The genealogic chart with the blood biochemistry findings demonstrates that the defect in carbohydrate metabolism, in one or both of the parents, or with each parent contributing a part of the deficiency, is what is actually transmitted as the heritage of deafness; namely, in most instances, the young patient exhibits a hyperpyruvemia and a hyper-

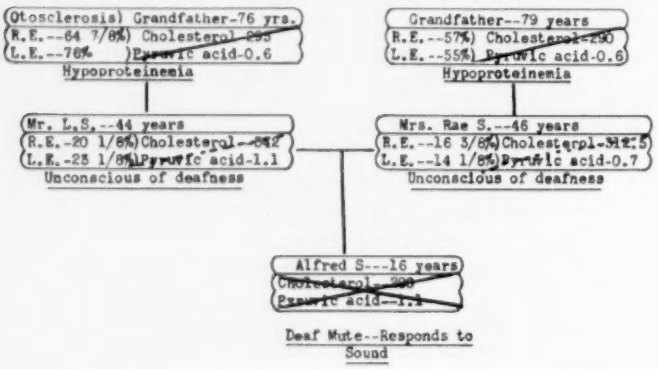

Fig. 9.—Blood lines of Alfred S., aged 16. A deaf-mute, he inherits from both sides, from the grandfathers on both male and female sides of his family tree. Both parents have a degree of deafness but are not conscious of it. The grandfathers on both sides of the family are deafened and conscious of it. Both parents as well as the boy carry hyperpyruvemia and hypercholesteremia in their blood; the grandfathers on both sides exhibit hypercholesteremia only and hypoproteinemia, a not unusual coincidental finding with hypercholesteremia. Crossed lines indicate deaf-mutism; unbroken line, loss of hearing, and broken line, loss of hearing but no consciousness of it (asymptomatic otosclerosis).

cholesteremia. If the heritage has become attenuated, only a hyperpyruvemia is exhibited.

Reported here is a study pertaining to clinical and pathologic manifestations in 55 deafened children, of whom 25 were deaf-mutes, with

11. Gray, A. H.: Pathological Changes in Auditory Nerve in Otosclerosis and Their Significance Clinically, Especially with Regard to Paracusis Willisii, Proc. Roy. Soc. Med. **25**:1286 (June) 1932; J. Laryng. & Otol. **47**:598 (Sept.) 1932; Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:652 (Dec. 25) 1932.

5 having only residual hearing. The majority of these children were less than 10 years of age.[12]

All the children in this study exhibited hyperpyruvemia without exception.[13] This condition signifies a defective mechanism in carbohydrate metabolism in the enzyme carboxylase.[14] This deficiency leaves cholinesterase out of control and destroys acetylcholine, the mediator of nervous action along the synapses of the acoustic nerve.

The factors at fault in the dysfunction in the chemical interplay of the enzymatic system and the extracellular tissue fluid reactions of the cells of both bone and of cartilage, which these factors throw out of balance, produce additional results that terminate in abnormal local conditions in the hearing apparatus. This disturbance in the balanced relation affects the minerals necessary for the maintenance of proper alkaline reserve. It also affects the calcium and potassium ions at nerve

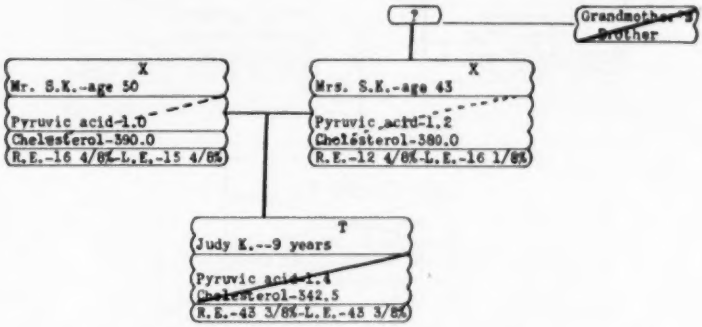

Fig. 10.—Blood lines of a 9 year old girl, who presents a great hearing loss. Both parents as well as the child exhibit hyperpyruvemia and hypercholesteremia. The parents have very slight hearing losses. The child's grandmother's brother was also deafened. The grandmother and her brother were not available for tests. Broken line indicates hearing loss but no consciousness of it; unbroken line indicates severe hearing loss. *X* indicates asymptomatic otosclerosis; *T* indicates symptomatic otosclerosis, presenting freely mobile ossicular chains but perception dysfunction type of deafness (first phase of otosclerosis).

endings, which are so necessary for the rapid functional development (synthesis) of acetylcholine and its ester, which are also put out of balanced reactions.

12. Of these patients, 23 acknowledged a familial history of deafness; 29 disclaimed any deafness in blood relatives, and 2 did not know whether or not any of their blood relatives were deafened.

13. Data from this study are presented in table 3.

14. Spies, T. D., and Butt, H. R., in Duncan, G. G.: Diseases of Metabolism: Detailed Methods of Diagnosis and Treatment, Philadelphia, W. B. Saunders Company, 1942.

Thus, on the one hand, the synthesis of acetylcholine is handicapped, and on the other, because cholinesterase is out of controlled physiologic function, acetylcholine, which might have been synthesized, is destroyed and the transfer of energy from a sound wave stimuli to a nervous impulse is interfered with at both the nerve endings and at its synapses along its tracts running centrally.

The clinical finding of failure in perception of an acoustic nerve (acoustic nerve type of deafness) becomes comprehensible. The clinical observation of the type of deafness in the early stages of otosclerosis has been made by many observers—Gray,[15] Holmgren [16] and others, including my own observation—and I, like Gray, believe that this

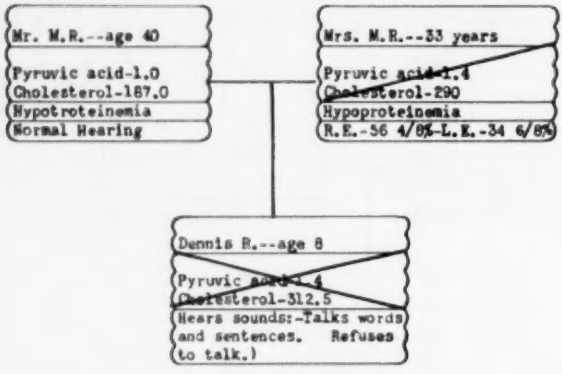

Fig. 11.—Blood lines of a child aged 8 apparently greatly deafened. Occasionally, he speaks short, intelligent sentences, suddenly and spontaneously; but generally he is uncooperative and refuses to talk. The diagnosis is audiomutism, secondary to severe hearing loss. The father exhibits hyperpyruvemia, a normal cholesterol range and hypoproteinemia. The mother exhibits both hyperpyruvemia and hypercholesteremia. The deafened son shows both hyperpyruvemia and hypercholesteremia.

The mother's medical history contains the story of a severe nervous breakdown. This history, considered in connection with my diagnosis of the child's type of mutism, is interesting because of the psychosomatic factors involved, which are presented in this child's case and which were uncovered at two examinations, separated by a few months. The absence of lines indicates good hearing; the unbroken line, severe deafness, and the crossed lines, deaf-mutism.

perception failure (nerve deafness) is found clinically long before stapes ankylosis is exhibited. Gray [15] went so far as to state that in his

15. Gray, A. H.: Otosclerosis: Its Treatment, J. Laryng. & Otol. **43**:21 (June) 1928. Footnote 11.

16. Holmgren, G., in Nelson Loose-Leaf Surgery of the Ear, edited by S. J. Kopetzky, ed. 2, New York, Thos. Nelson & Sons, 1947, p. 399.

opinion the type of deafness in otosclerosis which is known clinically through long periods of its course is due to the suspension of its function of hearing rather than to its actual destruction.

From my experience with the results in increased hearing acuity obtained by replacement therapy to supply the lack which the factors at fault in otosclerosis exhibit in the given case, there would appear to be a substantiation of Gray's contention. Otherwise it would be impossible to explain the halt of deterioration in hearing in so many cases and the gains of practical import in many others.[1]

In the otosclerotic patient, whether the given case of deafness is asymptomatic or symptomatic, there is presented a defect in phosphorylation mechanism of carbohydrate metabolism, particularly as it exhibits

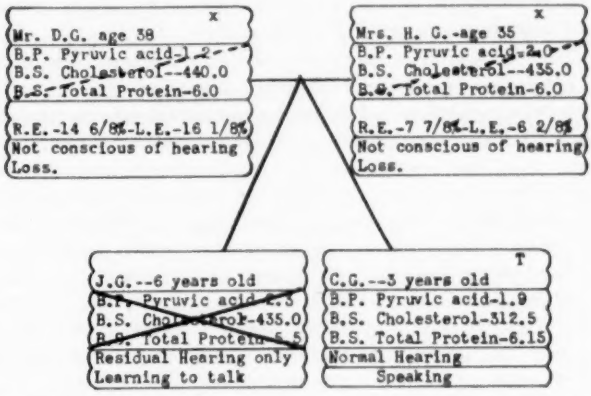

Fig. 12.—Case illustrative of how a brother or sister of a deaf-mute child can be, to all practical purposes, normal in speech development and hearing and nevertheless carry the factor at fault in otosclerosis, exhibited in their blood. The absence of lines indicates normal hearing; the broken line, slight hearing loss with no consciousness of deafness, and the crossed lines indicate deaf-mutism. *X* indicates asymptomatic deafness, and *T* indicates a sibling whose whole blood biochemistry demonstrates factors at fault in otosclerosis but who, at this time, both hears and speaks normally.

a deficiency in the diphosphothiamine-magnesium-protein item [14] of the vitamin B group. This condition deleteriously affects the health of endothelial tissue. The lining membranes of the arterioles and capillaries of the cochlear, and to a lesser degree also of the vestibular, apparatus are thus brought into the pathologic picture.

Their deterioration in health permits the second factor at fault, the hypercholesteremia, to produce an abnormal situation in the vascular channels. The hypercholesteremia in the otosclerotic patient and in his

children evidences a lack of lipotropic factors, which are the background, and the deposits of lipids in the arterioles and capillaries of the acoustic end organ occur for the following reasons: (1) the deterioration of the health of the cells in the lining of the vascular channels' endothelial tissue and (2) the resulting narrowing of the lumen of these blood vessels, with consequent anoxia of the area these vessels supply. Hence atrophic areas form as a consequence of this pathologic stage in the development of the lesions of otosclerosis. Otoscopically, this phase is evident, too, in the sluggish vascular reflex on the membrana tympani or in the absence of this reflex. I have observed this otoscopic phenomenon when the ossicular chain was freely movable and the eustachian tube fully patent.

In this regard Wittmaack's [17] experimentally produced blood stasis studies in his efforts to create artificially otosclerotic lesions in experimental animals are an interesting side study.

Finally, as seen in my biochemical studies of the blood, the defect in the carbohydrate metabolic mechanisms also affects the intermediate carbohydrate metabolism and throws the phosphorus-calcium ratio out of balance. There results abnormal osteoclastic and osteoblastic cellular activity with bone rarefication and newly formed bone abnormally placed in the layers of the otic capsule; or, a process of calcium leakage from areas on the otic capsule is seen; that is, areas are observed where halisteresis develops. Students of otic capsular pathology have observed both of these findings of types of bone rarefying mechanics in histopathologic sections. I believe the differing findings are to be explained by the one set of observers seeing osteoblastic and osteoclastic cell activity and the others seeing only the rarefied areas and very little osteoblastic and osteoclastic cell activity in their sections; the latter were observing a quiescent lesion while the former were observing an active one. It seems improbable to assume a leakage of calcium without the cellular activity which both removes calcium and deposits it playing its wonted role through the osteoclasts and the osteoblasts.

Thus, from the findings of the blood biochemistry it is possible to comprehend the significance of what is happening in the living tissues of the otosclerotic patient in that final phase of otosclerosis when newly deposited bone impedes the labyrinthine windows and causes fixation of the ossicular chain and gives its dominant clinical evidence of a raised lower tone level above 64 double vibrations, with prolonged bone conduction over air conduction obtained by functional tests in a case

17. Wittmaack, K.: Die Otosclerose Aufgrund eigener Forschungen, Jena, G. Fischer, 1919.

in which a family history of deafness is presented. These three factors are the clinical criteria to establish a diagnosis of a fully developed lesion of otosclerosis.

These three pertinent factors which heretofore have been held as the criteria on which to base a diagnosis of otosclerosis are the Bezold triad, but when there is denial of a history of deafness in the blood lines of the patient, this triad cannot be obtained.

Now, however, by making biochemical examinations of the blood instead of relying on a patient's knowledge of his family's medical history, it is possible to reestablish a triad, totally irrespective of anything the patient tells of his family. Following is the suggested triad: (1) the finding of the factors at fault—hyperpyruvemia only or in conjunction with hypercholesteremia; (2) prolonged bone conduction over air conduction, and (3) a raised lower tone limit above the 64 double vibration level. With this triad presented, a diagnosis of otosclerosis is warranted.

Heretofore, this has been impossible because the relation of this biochemical abnormality in the blood to the cellular pathologic condition on the otic capsule was unknown, as was the relation of this biochemistry to the findings from otoscopy and functional tests.

One more item needs elucidation. Why does the cochlear nerve so readily fall into dysfunction because of environmental biochemical pathologic changes, whereas other cranial nerves seemingly are exempt from being similarly affected? The acoustic branch of the eighth cranial nerve, particularly its middle and terminal regions, possesses the lowest resistance to deteriorative environmental elements and diseases of any cranial nerve.[18] Compared to the resistance offered by the facial nerve to detrimental environmental influences, the difference is very marked. In this connection one needs but to reread the debates on the effects of arsphenamine on the cochlear and vestibular nerves,[19] and the effects of syphilitic infections which, sometimes as early as seven days after infection, resulted in deaf-mutism, as happened in a case reported by Politzer. Finally, as shown by recent experiences, when streptomycin is used in high dosage and the body does not excrete it adequately, there results a general toxicity with an especially deteriorative effect on the auditory and vestibular mechanisms.[20]

18. Alexander, G.: Pathologische Anatomie der nervöse Anteile des Gehörorgans, in Alexander, G., and Marburg, O.: Handbuch des Neurolgie des Ohres, Berlin, Urban & Schwarzenburg, 1923, pp. 714-719.

19. Beck, O.: Syphiles des Ohres und seiner zentralen Bahnen, in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen-, Ohrenheilkunde, mit Einschluss der Grenzgebiete, Berlin, Springer-Verlag, 1926, p. 656.

20. Fowler, E. P., Jr., and Feind, C. R.: Toxicity of Streptomycin for the Auditory and Vestibular Mechanisms, Am. Rev. Tuberc. **60**:39 (July) 1949.

Alexander [15] stated the belief that the difference in the powers of resistance to deteriorative environmental factors between the facial and the other motor cranial nerves and the eighth cranial nerve stems from the fact that the cochlear apparatus appeared only in the ages when the amphibian animals and the birds evolved. The cochlear apparatus is thus the last phylogenic development in man and therefore the least stable and least resistant to environmental deteriorative factors, whether these be the viruses of syphilis or the toxins from chemical processes or from disorganized enzymatic processes diverted by these inherent chemical reactions to produce abnormal conditions, both within the cells and in the extracellular fluid surrounding the cells of the cochlear apparatus.

THERAPY

At the present state of knowledge, it may not, at this time, be possible therapeutically to remedy the defect in carbohydrate metabolic mechanisms which produces the deficiency of the diphosphothiamine-magnesium-protein group of the vitamin B complex. Because replacement therapy has greatly improved conditions of auditory dysfunction in adult cases, evidence is not presented that a replacement therapy would lead to an inherited body without such defect, particularly when such defect has been transmitted through a number of generations of a family's blood line.

On the other hand, even a replacement therapeutic remedy which supplies to the youngster's body that which it lacks for normal physiologic function of the auditory apparatus and which will hold the vascular channels free from obstructing lipid, prevent the narrowing of the lumen of the small arterioles and capillaries and keep endothelial tissue healthy may, in the long run, keep otosclerotic cellular abnormalities in abeyance for a long enough time to be considered of practical usefulness. The replacement therapy must aim to supply the factors which will aid the transference of the energy of sound waves to acoustic nerve impulses.

Since such therapy is available, it can be instituted soon after the birth of a child, during the first year, at which time the cochlear and the vestibular apparatus develop and assume their wonted functions. At this time cortical brain centers gradually become functionally active also, so that replacement therapy for the child with a heritage of deafness can help him to appreciate sound symbols at a time when it is so very necessary to accumulate sound symbol memories and speech experiences and to use the imitative faculty by which the child, during this first year of life, begins to learn words and their meanings and begins to repeat them. Likewise, the ability to stand erect, with con-

trolled equilibrium, is begun to be established, particularly the ability to hold the head erect and to move it freely under the control of the infants' will power. These children, whose parents carry hyperpyruvemia alone or hyperpyruvemia and hypercholesteremia in their blood specimens, should have a biochemical examination of the blood at the end of the first year of life to determine what their blood exhibits, and then replacement therapy should be started if indicated.

Children with large hearing losses, but not deaf-mutes, will also benefit by replacement therapy and educational programs carried out with the help of hearing aid amplified sound. The blood studies here too will be of help in determining what needs replacement.

Children with apparently normal hearing who have one or two deaf-mute siblings should undergo biochemical examinations of the blood to determine whether or not they also carry the heritage of otosclerotic deafness which would need only an intercurrent irritant factor to start a cellular abnormality into activity on the otic capsule. If they are found to carry the heritage, then replacement therapy is indicated as a preventive measure, because at this time in their young lives they have apparently normal hearing.

Young married couples who know that one or both have a family history of deafness, despite the fact that they are not conscious of having any hearing loss, should have a biochemical examination of their blood made before starting a pregnancy to eliminate, if possible, such of the factors at fault as their blood examinations exhibit or, at any rate, to acquire positive knowledge of whether they escaped the family heritage.

It is too early at this time to postulate any theory as to whether or not the further transmission of the defective metabolic mechanism can be stopped by replacement therapy.

The term replacement therapy has been used here. Therapy for the deafened will be discussed in a later report.

SUMMARY

The heritage of deafness is reported in a study (table 3) of 25 deaf-mutes, 5 severely deafened children and 25 children with serviceable hearing, but deafened. The majority of these children were less than 10 years of age. The relations between the biochemical abnormalities in the blood, the symptomatology in its successive phases of otosclerotic evolution and the cellular abnormality on the otic capsule in otosclerosis were established.

The criteria for establishing a diagnosis of asymptomatic otosclerosis in the living patient is presented, based on blood biochemistry found in the familial blood lines of the patients' parents and, in some cases,

also the grandparents of deaf-mute children. These findings negate the accepted concept of skipped generations in the heritage of deafness.

The congenitally deafened have carbohydrate metabolic mechanism defects transmitted to them which develop otosclerotic lesions on the otic capsule in intrauterine life, or they carry in their blood the biochemical pathologic changes of otosclerosis which develop lesions after birth, owing to the immediate etiologic role which an intercurrent irritant factor induces.

The heritage of otosclerotic deafness is a defect which interrupts the physiologic interplay of the chemical processes of the enzymatic systems, secondary to a defective phosphorylation mechanism of carbohydrate metabolism, of which the diphosphothiamine-magnesium-protein group of the vitamin B complex is one of the principal deficiency items.

Deafness due to acoustic nerve dysfunction (perception failure) precedes the conduction interference with sound wave transmission, because of ossicular chain immobility or lack of elasticity of the membranes of the labyrinthine windows.

The blood biochemistry gives a rational explanation as to why the cellular pathologic changes of the otic capsule must result from what the biochemical pathology exhibits as an abnormal process in the different developmental phases of the lesions of otosclerosis.

Heretofore this explanation was impossible, although the evolution of the pathologic picture in each of its developmental phases has been known. Also known, to both biochemists and physiologists, are the biochemical reactions in the various portions of the enzymatic systems and the processes which control the synthesis and resynthesization after decomposition, through the activity of the carboxylase to produce the wonted effects on acetycholine. But their very close interrelation to the clinical exhibition of symptoms from the hearing apparatus and the results which follow when these reactions are diverted from their physiologic role and are forced, as a biochemical reaction, purposely to produce abnormalities which result in cellular pathologic changes of the otic capsule, producing the lesions of asymptomatic and symptomatic otosclerosis, have not heretofore been postulated.

A substitute for the Bezold triad, as diagnostic data, has been suggested, a triad which is not dependent on any statement of the patient or the patients' parents as to the presence or absence of a history of deafness in the family.

The replacement therapy to supply the essential items which hyperpyruvemia alone or hyperpyruvemia and hypercholesteremia indicate as needed has been tentatively discussed. A special report will be made on the therapy of otosclerosis.

TABLE 3.—*Summary of Data on Fifty-Five Deafened Children*

Case	Item		Father		Mother	
	Cholesterol	Pyruvic Acid	Cholesterol	Pyruvic Acid	Cholesterol	Pyruvic Acid
1	140	0.7	.....	...	.....	...
2	250	0.9	410.0	0.9	215.0	0.9
3	200	0.8	.....	...	.....	...
4	280	0.856	.....	...	.....	...
X 1	175	0.8	.....	...	.....	...
X 2	180	0.8	.....	...	.....	...
5	198.0	0.840	.....	...	.....	...
6	167.5	0.826	.....	...	.....	...
X 3	207.5	0.826	.....	...	.....	...
X 4	237.5	0.840	.....	...	.....	...
7	265.0	0.970	.....	...	.....	...
8	280.0	1.0	250.	1.0	290.0	0.8
X 5	175	0.8	.....	...	.....	...
9	187	0.826	.....	...	.....	...
10	187	0.84	.....	...	.....	...
11	185	0.84	.....	...	.....	...
12	175	0.80	.....	...	.....	...
X 6	330	0.616	.....	...	.....	...
13	237.5	0.813	.....	...	.....	...
X 7	155.0	0.970	.....	...	.....	...
14	155.0	0.853	.....	...	.....	...
15	250.	0.793	187	1.0	290.0	1.4
16	167.5	0.80	.....	...	.....	...
17	177.5	0.783	.....	...	.....	...
18	187	0.826	.....	...	.....	...
19	280	1.55	195.0	0.7	207.5	0.8
20	305 / 187	0.813	.....	...	280.0	0.8
x 8	250 / 195	0.8	.....	...	.....	...
21	330	0.8	.....	...	.....	...
22	195	0.826	.....	...	.....	...
23	250	0.813	.....	...	.....	...
x 9	312.5	0.816	.....	...	.....	...
x10	222.5	0.786	.....	...	.....	...
24	250	0.840	.....	...	.....	...
25	187.0	.80	.....	...	.....	...
x11	200	0.743	.....	...	.....	...
26	213.0	0.9	222.5	1.2	160.0	0.8
27	175.0	2.1	305.0	0.6	185.0	1.2
28	187	1.5	157.5	0.8	175.0	1.0
29	185	1.2	.....	...	.....	...
30	342.5	1.4	390.0	1.0	380.0	1.2
y13	209	1.12	227	1.37	209	0.6
y30	231	1.40	227	1.40	194	0.9
y31	158	1.14	160	0.74	221	0.69
y32	209	1.28	171	0.98	.....	...
y33	87	0.78	129	1.01	122	1.21
y34	153	?	129	1.01	122	1.31
xy35	282	1.38	178	1.43	253	1.23
y36	174	2.03	178	1.43	253	1.23
xy37	265	2.33	265	1.60	265	0.80
y38	178	1.10	265	1.60	265	0.80
y39	169	0.74	265	1.60	265	0.80
xy40	227	0.72	174.0	0.88	143	1.27
xy41	Deaf-mute, died at age of 5		297.0	1.71	228	0.69
31	435.0	2.3	440.0	1.2	435.0	2.0

Dr. Murray Abrams, a member of the staff at the New York Polyclinic Medical School and Hospital, a consultant in otology to the New York Public School System (assigned to P. S. 47, the School for Deafened Children) and assistant surgeon on the otolaryngologic staff of Bellevue Hospital, examined a group of deafened and deaf-mute children and supervised the taking of blood specimens from them and their parents, at my direction. The material he submitted to me carries the identification y in the appended list of cases comprising this study (table 3).

The biochemical examinations of the blood of the group marked y were made under the supervision of Dr. Norman Malomut, biochemist.

In all other cases, the biochemical examination of the blood were performed under the supervision of Mr. Nicholas Molnar, B.S., M.S. and Fellow of the American Institute of Chemists.

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CHEMICAL BURNS OF THE ORAL CAVITY AND ESOPHAGUS

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CHICAGO

CHEMICAL burns of the oral cavity and esophagus are widespread in the southeastern European countries. This condition is especially prevalent in Hungary and the Balkan countries. In Western Europe there are fewer cases. From 1903 to 1923 Guisez in France had seen only 169 of such cases. The long fight led by Chevalier Jackson to secure the proper labels on containers of lye has greatly decreased the number of burns from lye and caustics in the United States.[1]

At the otolaryngologic clinic of the University of Sofia (Bulgaria) there were 1,221 cases of chemical burns for a period of about twenty years (1928 to 1947). At this clinic much time has been devoted to the study of the pathology of these burns and various methods of treatment have been applied. Lye was responsible for most of the chemical burns of the oral cavity and the esophagus—979 cases. If lye were eliminated from household use, chemical burns would be greatly decreased.

The other chemicals causing burns of the esophagus and oral cavity, according to the statistics of this clinic, are presented in the following tabulation.

Chemicals	Number of Cases
Hydrochloric acid	39
Nitric acid	62
Sulfuric acid	107
Ammonia	6
Saponated cresol solution	6
Tincture of iodine	2
Phenol	1
Potassium permanganate	2
Kerosene	1
Spirits of camphor	1
Various mixtures	13

Read before the Detroit Otolaryngological Society, Jan. 19, 1949.

*Now at the Passavant Memorial Hospital and Northwestern University Medical School.

1. Jackson C.: Bronchoscopy and Esophagoscopy: A Manual of Peroral Endoscopy and Laryngeal Surgery, ed. 2, Philadelphia, W. B. Saunders Company, 1927. Taylor, H. M.: A Preliminary Survey of the Effect Which Lye Legislation Has Had on the Incidence of Esophageal Stricture, Ann. Otol., Rhin. & Laryng. **44**:1157 (Dec.) 1935.

Of these 1,219 cases, the condition in 603 was acute and in 618 chronic and strictural.

Clinically, the burns can be classified into three groups, with regard to the type, amount and concentration of the ingested caustic.[2]

First degree burns were present in about 10 per cent of all the cases. The signs and symptoms are vomiting and some pain of the esophagus. The pulse is normal, and there may be a slight rise of temperature. Pathologically, the superficial epithelial cells slough out and there is an edema of the mucous membrane and the submucous layer, but the deeper layers are not affected. If the caustic reaches the stomach it may cause inflammation of that organ.

Second degree burns constituted about 63 per cent of all the burns. With these burns the lesions are severer and they bleed. Pain is so severe during swallowing that the patient cannot even swallow his saliva. Some parts of the mucous membrane slough out. Because of the severe pain no food can pass through the esophagus. On roentgenographic examination a narrow passage of the barium can be seen. Sometimes an infection may result, and the temperature may rise to 102 to 104 F. After several days there is an improvement in the symptoms—the pains decrease and the temperature drops to normal. A state of well-being sets in for the next eighteen to twenty-five days, but it ends with the onset of stenosis of the scarred area, usually in four to six weeks.

The third degree burns, which occur in the severest cases and in those with eventual death, were caused by the ingestion of highly concentrated caustic solutions. These cases constituted about 27 per cent. In burns caused by acids the mucous membrane from the lips down to the stomach is the color of black coffee. In burns from alkaline solutions, such as lye, the mucosa is severely swollen and the ulcers are covered with a brownish gray exudate or slough. In such cases there is necrosis not only of the superficial layers but also of the deep layers, the muscular layer and the periesophageal tissue. This burned mucosa is sometimes eliminated like a glove as a vomitus.

In patients with this condition the face is drawn and cyanotic; the limbs are cold and also cyanotic; the pulse is weak, and respiration is accelerated and accompanied, usually, with high temperature. The patient is very thirsty and has severe pain and continual vomiting, which lasts about one or two days, and the patient dies from heart failure and general intoxication.

2. Belinoff, S.: Treatment of Corrosive Esophagitis, in Terracol, J. and others: Les maladies de l'œsophage, Paris, Masson & Cie, 1938.

Pathologically, there are four stages in cases of second degree burns.

The first stage is that of hyperemia and edema of the mucous membrane. This edema is caused by the absorption of the caustic solution by the cells and as a result of the reaction of the cells. In the very light cases there is only a sloughing out of the superficial epithelial cells. The mucosa is frequently eroded.

The first stage passes into ulceration, the second stage. After the exudate or slough falls out, the ulcers appear. The surface of the ulcers is irregular, covered with a yellowish or fibrohemorrhagic exudate, and is of various depths.

The third stage occurs about the third week. During this stage the ulcers are covered with granulation tissue. Later, these granulations are replaced by fibrous tissue. During this granulation stage the patient no longer complains of dysphagia. He can swallow without any difficulty, and he considers himself cured. But, unfortunately, this period lasts only a short time. After about four to eight weeks the patient comes back with stricture of the esophagus and dysphagia.

During the fourth stage stricture of the esophagus occurs. The granulation tissue is gradually replaced by the fibrous tissue, which forms the primary stricture.

The strictures may be single at one level or multiple at two or more levels. Later strictures of the esophagus as a result of corrosion from caustics are most frequently found at the level of the anatomic or physiologic narrowings of the esophagus. According to the statistics presented, they occurred first, in point of frequency, in the middle third of the esophagus or at the level of the crossing of the left bronchus in about 45 per cent of the cases; in the upper third of the esophagus or at the region of the cricopharyngeus in 42 per cent, and, next, in the lower third or at the hiatal level in 13 per cent. These findings are in accordance with those of Jackson and Hacker.

Sometimes there are total atresia of the esophagus and retention and regurgitation of all food ingested by mouth. The patient drools, loses weight rapidly, becomes acidotic and dehydrated, and if gastrostomy is not done death follows as a result of starvation.

In the differential diagnosis, one must differentiate cancer, aneurysm with compression, cardiospasm and presence of foreign body and diverticulum from benign stricture due to chemicals. Typhoid, scarlet fever and diphtheria may also cause fibrotic strictures. In most cases the diagnosis of cicatricial strictures of the esophagus and their cause can be determined from the history given by the patient. But roentgenography and esophagoscopy are necessary to arrive at a diagnosis.

and, especially with use of esophagoscopy, a positive result is always possible.

Esophagoscopy can be performed during the first stage of the disease without danger, for during this stage the wall of the esophagus is covered with a thick membrane and perforation of the esophagus is difficult. During the second and third stages, because of the presence of ulcerations and granulations, esophagoscopy is not advisable. If it is considered very necessary, it must be done with great care, because after the sloughing out of the necrotic parts the wall of the esophagus gets thinner and perforation of the wall is possible, even in the hands of the best esophagoscopist. Therefore esophagoscopy must be avoided during the second and third stages of the disease, unless there is a very urgent indication for the procedure.

The complications of corrosive esophagitis are numerous. The resulting complications, according to the statistics compiled from these cases, are presented in the following tabulation.

Condition	Per Cent
Mediastinitis	20
Perforation of the esophagus	15
Perforation of the stomach	10
Peritonitis	15
Edema of the larynx	6
Bronchopneumonia	14
Ulceration into the aorta or into the trachea	20

If the esophagus is perforated, the patient experiences back pain and has difficulty in breathing, a high temperature and septic symptoms, such as chills, a rapid pulse rate and weakness. After each perforation there is inflammation of the periesophageal tissue—mediastinitis. Mediastinitis is not always fatal. If adequate treatment is used the infection can be cleared. No food or liquids should be given by mouth, and penicillin and the sulfonamides should be given. The perforation of the esophagus is caused in some cases by the ingested caustic material and in other cases by the bougie during dilation.

TREATMENT

There is a standard course of action to be followed with each burn in order to prevent atresia.

The emergency treatment in the acute case is the prompt lavage of the mouth, esophagus and stomach with the proper neutralizing solution. It is possible to neutralize the offending agent in the first three or four hours, except when the caustic solution is very concentrated and penetrates the tissue very rapidly.

Caustic alkalis can be neutralized with vinegar, tartaric acid or lemon juice.[3] Olive oil is also given.[4] Caustic acids can be neutralized best with magnesium oxide. Milk should be given. Sodium bicarbonate should be avoided because it forms gas. Water is allowed the first day, and in cases of burn with caustic alkalis, such as lye, for instance, olive oil is given as food. In cases with dysphagia fluids should be given parenterally. If the patient cannot swallow during the following days, gastrostomy is indicated.

Livshits advised the use of short wave diathermy and roentgenologic treatment. This treatment reduces the pain, acts as an antispasmodic, is bactericidal and desensitizing, regulates the vagosympathetic tonus and increases phagocytosis. The short wave diathermy has been applied at the clinic only, and the results have been satisfactory.

After the formation of stricture, which occurs about the third and sixth week, dilation should be started as soon as possible.

There are different methods of dilation. Following are the methods applied in the clinic: The first or the very light burns are treated symptomatically. Rarely does a stricture form in these cases, and in some cases dilation is necessary.

It is mostly the patients with second degree burns who come for dilation treatment.

Of the treatments used, peroral esophagoscopic bougienage is the best and safest in cases in which gastrostomy is not done. For this purpose the flexible, semisoft fiber bougies are used. In 270 of the cases this method of treatment was used. Of these cases the condition in 20 cases completely healed, and there was improvement in 235, no improvement in 10 and death in 5. The size of the bougie is determined by the resistance to passage through the area of stricture.[5]

The bougie, introduced through the esophagoscope, is left in the esophagus for about thirty minutes, and this procedure can be repeated every other day by increasing the number of the bougie, reaching number 45 French.

Salzer recommended the use of rubber bougies filled with mercury or lead shot. With this type of bougie pressure is not applied, and this is blind bougienage. There is no danger of perforation because the bougie advances downward by the aid of its own weight. Treatment with the bougie of Salzer was applied to 39 patients, of whom

3. Terracol, J., and others: Les maladies de l'œsophage, Paris, Masson & Cie, 1938.

4. Vinson, P. P.: Management of Benign Stricture, J. A. M. A. **113**:2128 (Dec. 9) 1939.

5. Delph, J. F.: Chemical Burns of the Oral Cavity and Esophagus, S. Clin. North America **17**:585 (April) 1937.

9 were healed and 30 improved. Since 1935 this treatment has been abandoned.

There are two schools of thought concerning the time for beginning the dilation of the esophagus.[6] The one headed by Salzer,[7] of Vienna, advocated that dilation be instituted immediately after the burn. However, it was found that the reaction of a patient's acute burn to bougienage was quite severe, as evidenced by pain, fever and bleeding.

The other school of thought, headed by Guisez, of Paris, maintained that bougienage should be instituted at the end of the third week, after the acute swelling has subsided and granulation tissue has covered the ulcerations.

In the Sofia clinic, the method of Guisez was applied, and the results were satisfactory.

Another method which was applied and later abandoned, was that of use of the Nelaton balloon. This apparatus is composed of a urethral catheter, at the end of which is attached a balloon. The urethral catheter, with the aid of the esophagoscope, is introduced into the esophagus to the cardia. The balloon is insufflated and pulled up to the level of the stricture, where it is left for about five minutes. It is pushed down again, insufflated and then pulled up for a second and third dilation. Twenty-one patients were treated by this method; 5 recovered, 10 were improved, 5 were not improved and 1 died. Since the results were not satisfactory, this method was abandoned.

Another method was that of attempting to leave a permanent dilator in the esophagus. At the level of the sixth cervical vertebra, external esophagostomy was performed and the bougie was introduced through the esophageal opening. Owing to the high percentage of mortality, this method was also abandoned.

One of the best methods of treatment for stricture of the esophagus is the continuous string method.[8] Gastrostomy should be done with due consideration of its type and location for future retrograde bougienage.[9] When the gastrostomy wound has healed—that is, after

6. Jones, D. H.: Treatment of Lye Stricture of the Esophagus, Laryngoscope **55**:113 (March) 1945.

7. Salzer, H.: Treatment of Burns of the Esophagus, Wien. med. Wchnschr. **77**:488 (April 9) 1927.

8. Woodward, F. D.: Complete Cicatricial Stenosis of the Esophagus, Ann. Otol., Rhin. & Laryng. **51**:94 (March) 1942. Tucker, G.: Cicatricial Stenosis of the Esophagus with Particular Reference to Treatment by Continuous String, Retrograde Bouginage with the Author's Bougie, Ann. Otol., Rhin. & Laryng. **33**:1180 (Dec.) 1924.

9. Kearney, H. L.: Unusual Cases of Cicatricial Stricture of the Esophagus, Ann. Otol., Rhin. & Laryng. **44**:527 (June) 1935.

fifteen to twenty days—an attempt is made to have the patient swallow a silk string. The string should not be very long, so that it may not pass down to the intestines. Swallowing water helps the string to go down. After about twenty-four to forty-eight hours, the free end of the string is fished from the stomach through the gastrostomy opening. A urethral bougie is attached to the string, and by the upper end of the string being pulled, the bougie enters the stricture.

The length and thickness of the bougie used should be in proportion to the form of the stricture so that it may enter easily. To the other end of the bougie another string is attached, which remains outside the gastrostomy opening. The stricture is under constant dilation by the bougie, which stays in the esophagus for months and years. When the stricture is very narrow the patient can be fed through the gastrostomy opening. Later the patient can be fed by mouth. The food passes down either side of the bougie or through its lumen. When food passes with difficulty the patient is instructed to pull the bougie down into the stomach during meals, and after meals he pulls it up again.

For satisfactory dilation bougies no. 28 and 30 are used. Such dilation can be attained in one year or one and a half years, but the desirable period of dilation is two years, and six months after that the gastrostomy opening may be closed. Following are the advantages of this method:

1. There is no danger of perforating the esophagus.
2. The stricture is being dilated constantly.
3. It is painless and the patient can bear it.
4. The patient can continue the treatment without the aid of the physician.

I wish to point out that this method is the best so far and that good results were had in the clinic. Of the 156 patients treated with this method, the condition healed in 31, improved in 120 and did not improve in 3. There were 2 deaths from causes not related to this treatment.

In cases of complete atresia in which dilation is impossible gastrostomy is done and the patient is fed through the gastrostomy opening for the rest of his life. Such patients can live for many years and can return to gainful occupations. In other cases esophagoplasty is performed. The basis of this operation is the utilization of a cervical skin flap inverted on itself, into tubular form, with the skin inward. In other cases a part of the small intestine is taken and implanted under the skin of the chest; this can satisfactorily substitute for the esophagus, with peristaltic movements.

SUMMARY AND CONCLUSIONS

Chemical burns of the oral cavity and esophagus are classified, pathologically and clinically, into three groups, with regard to the type, amount and concentration of the ingested caustic.

It is mostly the second degree burns that need a systematic treatment for the prevention of stenosis of the esophagus.

The emergency treatment should be applied as soon as possible. After formation of stricture, dilation should be started immediately.

Esophagoscopic bougienage can be applied in cases with a slight stricture.

The continuous string method is the best in cases with a marked stricture. In this kind of treatment the bougie should be left in the strictural area constantly till satisfactory results have been obtained.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

THE PARANASAL SINUSES

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ANATOMIC STUDIES

WELLS [1] gives a historical sketch of Nathaniel Highmore, who first described the maxillary sinus in 1651, his interest having been aroused by a case of suppuration of dental origin with drainage through the tooth socket.

Marchi [2] sectioned 71 heads of mammals obtained from the Zoological Institute of Siena and the Craniological Museum of the University of Siena and studied the conformation and relationship of the sinuses in cattle, horses, sheep, dogs, cats and monkeys. An interesting observation in many of these animals is the existence of accessory cavities, such as palatine, oral and aboral, communicating directly with the maxillary, frontal and sphenoid sinuses. In fact, it appears that all the sinuses communicate with each other in chain fashion, the frontal with the maxillary, the maxillary with the palatine and the palatine with the sphenoid. The analogy to this in the human being is to be seen in the form of a frontomaxillary canal, as described by Fiol, which is particularly developed in the horse. In the human being it is often observed to be well developed, offering a direct pathway from the frontal to the maxillary sinuses, terminating in an oval aperture of varying dimensions. Some importance is attached to this conformation in the explanation of chronicity in certain infections and simultaneous involvement of the two cavities. Several roentgenograms of the sectioned skulls show a remarkable degree of pneumatization in some of the higher primates.

Haley [3] presents his observations on 35 human skulls, showing the variations in size and extent of the various sinuses. A number of illustrations of sections reveal some unusual variations. Several of the items

1. Wells, W. A.: Nathaniel Highmore, Seventeenth Century Pioneer in Anatomy and Embryology, 1613-1685, Laryngoscope **58**:583 (June) 1948.

2. Marchi, R.: Sulla pneumatizzazione delle ossa facciali nei mammiferi e sull'esistenza del canale maxillo-frontal di Fiol, Arch. ital. di otol. **57**:178 (July) 1946.

3. Haley, J. C.: A Study of the Extent, with Variations, of the Bony Framework of the Paranasal Sinuses, M. Rec. & Ann. **42**:693 (Dec.) 1948.

noted are (1) the projection of the infraorbital canals, often as much as 7 mm., at the junction of the roof and anterior wall; (2) the general asymmetry of the frontal sinuses; (3) the irregularity in the size of the sphenoid sinuses, with frequently prominent recesses into adjacent vital areas; (4) the wide variation in the number and size of the ethmoid cells.

Monti [4] sectioned a number of fetuses ranging from 11 to 37 cm. and studied the conformation of the lateral wall of the nose, with the idea of determining the embryologic origin of the maxillary sinus. He concludes that the early formation of the sinuses is not due to the invagination of nasal mucosa into the cartilaginous capsule of the primitive middle meatus, but rather to an invagination of the cartilage itself where it terminates below at the level of the inferior turbinate. Here it bends inward in a line with the upper border of the turbinate with which it is directly continuous. A reproduction of a wax model shows this clearly.

Jacobs [5] includes a number of illustrations depicting variations in the structure and outline of the maxillary sinus. It is important to recognize these aberrations when contemplating surgical treatment of the sinus. Not only is there considerable variation in the thickness of the bony walls and size of the sinus depending on hereditary influences, the presence or absence of teeth and the development of the alveolus, but there may be pockets or extensions of the sinus in various directions. Roentgenograms properly taken can demonstrate all these variations. Hence good pictures properly interpreted are invaluable.

PHYSIOLOGIC AND PATHOLOGIC STUDIES

By means of experiments on rabbits, Hilding [6] sought to obtain light on the question of the importance of ventilation of the sinuses. In a series of 5 animals, one nostril was hermetically sealed by means of a plastic closure and later the maxillary ostiums were enlarged. The 5 animals lived from one to two years. During that time twice the normal volume of air passed through the unobstructed nostril. In only 1 of the animals were the sinuses observed to be clean and free from infection. The other 4 acquired bilateral sinusitis, 2 spontaneously and 2 after enlargement of the maxillary ostiums. In 3 of them there was no difference between the two sides. From this small series of experiments it appears that ventilation alone does not prevent or cure sinusitis.

4. Monti, P. C.: Sullo sviluppo del seno mascellare, Ann. laring., otol. **45**:4, 1946.

5. Jacobs, M. H.: Anatomic Study of the Maxillary Sinus from the Standpoint of the Oral Surgeon, J. Oral Surg. **5**:282 (Oct.) 1947.

6. Hilding, A. H.: Experimental Sinus Surgery: Some Experiments on Ventilation and Sinusitis, Laryngoscope **58**:1098 (Oct.) 1948.

Tremble [7] studied the rate of ciliary movement by means of a blower through which a small amount of finely powdered dibasic calcium phosphate colored with a harmless orange dye could be deposited at any given point in the nose and its progress noted. His observations confirmed the results of several experiments previously reported by other authors and provided some additional new data. In the anterior third of the nasal chamber, where the first impact of inspired air is encountered, the secretion of mucus is less abundant than in posterior portions, being renewed in an hour or two. In the posterior two thirds of the cavity the new film of mucus is formed in ten minutes. Ciliary activity likewise is correspondingly slow and rapid according to the degree of exposure of the particular area to the inspiratory blast. Normally the mucus secreted is made up of 3 parts mucin, 2 parts salt and 95 parts water. If the proportion of mucin is increased, the mucous blanket is heavier and moves more slowly. Likewise, if the amount is decreased so that the water content is above normal, ciliary activity is slowed. Numerous other observations on the rate of ciliary activity are recorded in such conditions as atrophic rhinitis, acute inflammatory processes, allergy, polypi and septal deformities.

Van Dishoeck [8] offers some observations on the effect of nasal reduction on nasal respiration, citing a recent communication on this topic by Šercer.[9] The latter held that raising the tip of the nose improves breathing by favoring the passage of the air stream into the upper parts of the nose. Van Dishoeck claims that his observations in patients, as well as in experiments on models, prove that the air current "obeys a simple aerostatic law in following the way of least resistance." The curved form of the air stream is not "caused" by the form of the turbinates, but this semicircle represents the path of least resistance through a chamber with the inflow and outflow openings in the same—in cases, horizontal—plane, whether turbinates are present or not. A high curved air stream is not essential to the sense of smell; the olfactory organ being situated high up receives only a fraction of the inhaled air by way of small eddies ascending to equalize the negative pressure caused by the previous inspiration. This accounts for the persistence of the sensation after the inspiratory phase. Improved respiration following elevation of the tip is due not to alteration in the direction of the stream of air but to the widening of the internal ostium of the vestibule. The author considers this to be the important factor.

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7. Tremble, G. E.: Clinical Observations on the Movement of Nasal Cilia: An Experimental Study, Laryngoscope **58**:206 (March) 1948.

8. van Dishoeck, H. A. E.: Some Remarks on Nasal Physiology, Inspired by the Study of Šercer on Nose-Reduction, Acta oto-laryng. **36**:467, 1948.

9. Šercer, A.: L'inclinaison de l'orifice narinaire—comme cause de l'insuffisance respiratoire, Acta oto-laryng. **35**:565, 1947.

Colson [10] carried on several new experiments with various Venturi tubes with attached manometers in an attempt to elaborate further his theory of the efficacy of nose blowing in sinus drainage according to the Bernoulli principle. It appears that in most noses the conditions favoring the exemplification of this principle are present, namely, that the current of forcibly expelled air passing from the choanae through a constricted area in the vicinity of a sinus ostium induces a negative pressure at this point whereby secretions propelled to the ostium by the ciliary membrane within the sinus are expelled. This cannot occur, however, if the posterior choana is blocked by turbinal swelling, nor is it effective in grossly atrophic noses.

Cantor [11] conducted a number of experiments, with a simple glass tube apparatus simulating the maxillary sinus, in an attempt to explain the expulsion of secretions following nasal irrigation with warm saline solution.

Contrary to the belief that the discharge is due to the suction effect of the irrigating fluid flowing over the sinus ostium, he shows that the sinus secretion is actually propelled from the sinus by increased air pressure within the sinus, the air expanding under the influence of the heat transmitted by the warm irrigating fluid. Various mathematical calculations are produced explaining the phenomenon on a physical basis.

Oliveira Lima [12] describes the histologic changes observed in the nasal and sinus mucosa resulting from allergy. These vary according to the degree of sensitivity of the tissues, the potency of the allergen and the duration of the process. In acute conditions the characteristic observations are edema and infiltration with eosinophils. Chronic disorders are characterized by involvement of all the tissues, including the bone. The pathologic process may eventually become irreversible. This is more likely to occur in the perennial than in the seasonal types because of prolonged stasis, damage to the blood and lymph vessels and the secretory apparatus and intercurrent bacterial infection. The author presents in detail an analysis of the changes in the epithelium, basilar membrane, tunica propria, mucous glands, blood vessels and osteoperiosteum. Edema is likely to be most pronounced in the tissues having little stroma and is influenced by gravity and the conformation of the mucosa, which accounts for greater development in the ethmoid cells and maxillary sinus.

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10. Colson, Z. W.: Nasal Sinus Drainage: Further Bernoulli Experiments, Laryngoscope **57**:642 (July) 1948.

11. Cantor, J. J.: The Removal of Secretions from the Nasal Antrum, Laryngoscope **58**:1307 (Dec.) 1948.

12. Oliveira Lima, A.: Histopatologia da mucosa nasosinusal nas inflamacões alérgicas, Brasil-med. **62**:142 (March 20-27) 1948.

DIAGNOSIS

In an excellent monograph by Buch [13] an attempt is made to answer the question of the incidence of undiagnosed sinusitis. It is based on routine examination of 4,682 patients not suspected of having sinusitis who were under observation for other ailments. The examination consisted of an analysis of symptoms that might be referable to the sinuses, roentgenologic studies and, when indicated, exploratory puncture and lavage. The over-all incidence in all groups was 8 per cent, but in patients being treated for diseases of the respiratory organs and the ear the percentages were 20 and 13. The lowest incidence of sinusitis was encountered in patients receiving treatment for diseases of the kidney and urinary tract. The author refuses to consider the group of patients with disorders of the circulatory system, among whom the percentage of cases of sinusitis reached 13, because the number of persons involved was too small in comparison with that in the other groups. Another item of interest was the fact that the incidence in persons beyond the age of 45 was double that in persons in the lower age brackets.

The reasons the sinusitis was not diagnosed were as follows: In 25 per cent there were no subjective symptoms. In 32 per cent the symptoms present were attributed to the other disorders. In 41 per cent the symptoms could have been attributed to the sinuses, but in most patients were ignored by either the patient or the physician.

Characteristically, the absence of pain was considered an important factor in the neglect, whereas the commoner symptoms of obstruction and discharge were ignored. Items such as the value of roentgenograms and lavage in diagnosis are also thoroughly gone into. It was established that roentgenograms showing clearness of the maxillary sinuses could be depended on in 99 per cent of cases as indicating absence of infection. On the other hand, pictures showing a cloudy sinus were seldom in error, as proved by irrigation and culture, as well as by biopsy. In cases in which the cloudiness did not indicate sinusitis it was shown to be due to anatomic anomalies, such as smallness of the sinus compared with the opposite one, shallowness of the sinus or the presence of septums.

Continuing the analysis in another contribution, Buch [14] makes some interesting observations based on a study of roentgenograms in this series of 4,682 patients in whom sinusitis was not suspected. Eight per cent of these revealed an opacity of one or both maxillary sinuses of various degrees. In the latter group, the antrums of 192 were punctured and lavaged; 10 per cent were normal, 65 per cent showed secretion and 25

13. Buch, A.: Non-Diagnosed Maxillary Sinusitis, Acta oto-laryng., 1949, supp. 77, p. 1.

14. Buch, A.: On Occurrence of Non-Diagnosed Maxillary Sinusitis, Acta oto-laryng., 1948, supp. 74, p. 303.

per cent mucosal thickening without secretion. The opacity noted in the normal maxillary sinuses was explained by the small size and relative shallowness of the cavity. In 744 persons with maxillary sinusitis referred by internists, 37 per cent complained of headache, 32 per cent had nasal symptoms and 31 per cent were referred because of otitic symptoms or signs of disease of the lower respiratory tract. In 25 per cent of the patients no symptoms were present. "The non-diagnosed cases of maxillary sinusitis accompanied by symptoms appeared to be of a markedly more chronic type than those of sinusitis which came spontaneously for treatment."

Another observation was the persistence of cloudiness of the maxillary sinuses for weeks or months following an attack of acute sinusitis. Patients showing sinusitis after routine examination but having no subjective symptoms were in the main disinclined to submit to therapy; 58 per cent of this group failed to return for treatment. Deviated septum was observed frequently in association with a cloudy antrum. Most of the 124 patients in whom resection was done showed clearing of the sinus following the operation. The 4,682 patients examined were divided into twenty-three groups according to a plan proposed by the Danish Board of Health. The group with diseases of the respiratory tract and the ear revealed a higher rate of opacity of the maxillary sinus than any of the other groups.

Cora Eliseht [15] insists that the contradictory concept of "sinusitis without sinusitis" should be dismissed from the minds of otolaryngologists. When a sinusitis exists, even with minimal symptoms, the diagnosis can be made if sufficient time and care are given to the problem. Roentgenograms frequently have to be repeated and studies with contrast mediums will reveal much that is pertinent, particularly if the emptying time is noted. Secretion obtained from the nasal cavities can yield valuable information, provided its source is accurately determined and such items noted as time of maximum flow, the area in which it was mostly observed, its amount, consistency, color and cellular content. He mentions such factors as congestive rhinitis and hyperplasia of the turbinal mucosa, which produce a secretion that might be taken to arise in the sinuses. He calls attention to the chronic pharyngitis and bronchitis frequently seen in association with chronic sinusitis in which the postnasal discharge is a factor.

The author has seen cases of lumbago [?] due to sinusitis, as well as of neuralgia in various locations, all of which he attributes to general toxemia. Such conditions can be relieved if the sinusitis is properly diagnosed and cared for.

15. Cora Eliseht, C.: Anotaciones clínicas de la infectión sinusal, Semana méd. **55**:412 (Aug. 26) 1948.

Larroudé [16] discusses the role of allergy in sinus conditions and offers a classification under the following heads: serous, catarrhal and purulent, the last named being divided into vegetant, polypoid and fibrous, depending on the dominant pathologic features. These various types are described, with accompanying illustrations showing the characteristic histologic appearance of the mucosa. It is important to differentiate an allergic swelling of the sinus mucosa from a hyperplastic condition, since the latter may frequently act as a focal nidus causing symptoms in other locations. The author relies greatly on the roentgenologic studies with contrast mediums, repeated if necessary, and with emphasis on the observation of the emptying time. He calls attention to the frequent association of allergy with infection, which increases the difficulty in diagnosis. In some instances it may be necessary to follow the usual diagnostic procedures with the taking of a biopsy specimen of the sinus mucosa through a trephine opening. This will provide material for not only a histologic study but also a cytologic study, both of which are important.

Fox [17] calls attention to the fact that many patients labor under the delusion that they are suffering from sinus disease, having come to this conclusion from self diagnosis based on the reading of lay literature or having been so informed by a physician not trained in otolaryngology. The symptoms on which this misinformation is based are usually headache, facial pains, nasal obstruction and postnasal discharge, each of which may be due to other causes. The author terms these cases "phantom" sinusitis or "pseudosinusitis" and mentions various conditions other than sinusitis which may cause the symptoms. Correct diagnosis depends on careful history taking, good general examination and proper evaluation of the findings.

Hauser [18] states that sinusitis is frequently diagnosed to cover symptoms of other origin. Nasal obstruction due to a deviated septum or to an allergy, postnasal discharge due to smoky environment or too free use of tobacco or alcohol and headaches due to ocular imbalance, metabolic disturbances or other disorders are all frequently mistakenly diagnosed as sinusitis. The taking of an adequate history and thorough examination should differentiate among them and lead to the proper therapy.

In cases of sinusitis in which there is doubt in the interpretation of cloudiness on transillumination or roentgenography, Frouchtman Roger

16. Larroudé, C.: Sinusites crónica e alergia, Gaz. méd. port. **1**:200, 1948.

17. Fox, C. C.: The Abused Diagnosis of Sinusitis, Pennsylvania M. J. **52**:136 (Nov.) 1948.

18. Hauser, I. J.: Diagnosis and Treatment of Sinusitis, Alexander Blain Hosp. Bull. **7**:76 (Aug.) 1948.

and Fernando Botet [19] advise the instillation of 0.5 cc. of epinephrine (adrenalin,* 1 in 1,000 solution) into the maxillary sinus with subsequent diaphanoscopy at intervals of a few minutes. They claim that if the process is solely an edematous reaction to an allergy the cloudiness will disappear in a short time. They report positive results in 4 of 8 cases in which it was tried. They also point out that other procedures are necessary for the diagnosis of an infective process superimposed on the allergy. No mention is made of cytologic investigation in these cases, although the work of several North American authors is cited.

Rhodes [20] emphasizes the importance of careful examination in the determination of respective roles of infection and allergy in the symptom complex. When allergy is the principal factor, surgical procedures on the nose should be avoided except for the necessary removal of polypi. Proper recognition of the offending allergens and their removal from the environment if possible, or treatment by desensitization, are discussed fully. The author does not favor the topical use of penicillin or the sulfonamide compounds.

Thoma [21] discusses involvement of the maxillary sinus from disease or lesions of the adjacent structures. The items covered are hematoma, infections of dental origin, cysts, benign tumors, locally infiltrating tumors and malignant tumors. The roentgenograms shown are especially instructive. Apical tooth infections are particularly likely to spread to the maxillary sinus through the intervening bone even in cases in which the distance is considerable. Trauma incident to extraction is a frequent cause. Roentgenologic studies are particularly helpful in the diagnosis. Among the benign tumors mentioned are osteoma, fibro-osteoma and odontoma. The infiltrating tumors are of the adamantinoma type.

Stahl [22] finds percussion of the maxillary sinuses a useful adjunct in diagnosis. It must be done with care in a quiet room. The middle finger of the right hand is used, striking the malar prominence at exactly the same point on both sides alternately with as nearly as possible the same stroke. One should be able to detect variations in pitch where the density differs on the two sides owing to swelling, fluid or tumor masses. The author claims this procedure is in the same class as transillumination, leading one to suspect an abnormality on one side when other

19. Frouchtman Roger, R., and Fernando Botet, J. M.: Contribución al diagnóstico de las sinopatías reaccionales, An. med., Barcelona **34**:261 (July) 1947.

20. Rhodes, W. L.: Differential Diagnosis and Treatment of Paranasal Sinusitis, M. Rec. **161**:98 (Feb.) 1948.

21. Thoma, K. H.: Diseases of the Maxillary Sinuses Resulting from Pathologic Changes, J. Oral Surg. **5**:271 (Oct.) 1947.

22. Stahl, R.: Zur Diagnostik der Kieferhöhlenerkrankungen (Perkussion der Kieferhöhlen), Deutsche med. Wchnschr. **73**:113 (March 12) 1948.

symptoms are minimal and pointing the way to further examination by means of contrast mediums and roentgenography.

Lemoyne [23] describes a method of puncturing the frontal sinus for diagnosis and therapy. He uses a short, heavy guarded trocar which is driven through the anterior plate by a few strokes of a mallet at a point 1 cm. above the intersection of a line running through the supraorbital margins and the midfrontal line. The procedure, he says, is applicable only in patients with large or moderately large sinuses and is done with the region under local anesthesia after some premedication. He does not advise it for patients with severe acute conditions or when complications are threatening. The puncture may be repeated if necessary until the penicillin which is instilled flows freely and rapidly into the nasal chamber and is recognized by the patient.

Leroux [24] followed Lemoine's technic of diagnostic puncture of the frontal sinus in 30 patients without any untoward results. He claims that the procedure is fully justified in patients in whom the frontal duct cannot be probed and whose symptoms and signs suggest involvement of the frontal sinus. Several patients were cured after a few washings through the puncture opening and several others were operated on because of the evidence disclosed by this method. The procedure is safe if the roentgenograms show the sinus to be of sufficient depth. The trocar cannot penetrate more than 8 to 10 mm. into the sinus.

Welin [25] describes the five standard projections employed at the Caroline Hospital, Stockholm, Sweden, in four of which the appearance of the frontal sinuses may be judged. These are (1) patient sitting with forehead and nose against the table, which is angled at 30 degrees from the vertical and leans against the patient's forehead; the central ray is directed horizontally toward the middle of the nose; (2) patient sitting with nose and open mouth pressed against the table, which is angled at 20 degrees from the vertical, with the central ray directed through upper lip; (3) direct lateral projection, patient either recumbent or sitting, with the central ray directed through the external orbital angle; (4) overshot axial projection, patient sitting or recumbent with head extended so that the mandible is projected in front of the frontal sinuses and the beam is tangential to their posterior walls; this position affords a clear estimate of the anteroposterior depth both medially and laterally, as well as the thickness of the walls; (5) patient positioned as for projection 4

23. Lemoyne, J.: La ponction du sinus frontal, Ann. d'oto-laryng. **64**:648 (Nov.-Dec.) 1947.

24. Leroux, L.: Résultats diagnostiques et thérapeutiques de la trépano-ponction du sinus frontal (technique Jacques Lemoine), Oto-rhino-laryng. internat. **32**:119 (July) 1948.

25. Welin, S.: The Röntgen Ray Examination of the Paranasal Sinuses with Particular Reference to the Frontal Sinuses, Brit. J. Radiol. **21**:431 (Sept.) 1948.

except that the head is not extended so far, the mandible being superimposed over the frontal sinuses; this projection shows the ethmoid sinuses to better advantage, particularly their anteroposterior and lateral extension.

The author shows several instructive roentgenograms, explaining the way a combination of the several projections can differentiate between local bony changes, swelling of the mucosa and retained fluid. One in particular is worthy of mention because the standard postero-anterior view showed an apparent absence of one frontal sinus, whereas the "overshot axial projection" revealed the presence of the sinus with severe osteosclerosis of the anterior wall. In special instances the author employs a sixth projection, in which the beam is directed as in projection 1, but with the patient lying in a true lateral position. This projection will often disclose fluid not observed in the other projections.

Anthony [26] reviews the roentgenologic procedures that have proved successful in the diagnosis of various lesions of the orbit and sinuses, with statistics showing the percentage of accuracy in the different groups. He discusses fractures, the optic foramen, inferior orbital fissure, exophthalmos, foreign bodies, nasal and facial fractures and diseases of the sinuses. He finds mucocele deceptive because instead of increased density there is a decrease due to pressure absorption of the bone. When associated with exophthalmos, considerable destruction of the normal bony landmarks may occur. Although mucocele is the commonest cause of exophthalmos, the author lists a series of at least twenty other lesions, most of which can show changes in the roentgenogram. Pictures of the sinuses are invaluable, especially when surgical treatment is contemplated, and the author points out some of the diagnostic points for which one should look.

McGovern, Fitzhugh and Cooper [27] describe a number of pathologic conditions seen in roentgenograms of the head which should interest the otolaryngologist. Frequently, in looking over the films taken primarily for diagnosis of sinus disease, one encounters abnormalities in the skull due to other causes, which may, however, account for the symptoms that prompted the roentgenologic examination. Among the conditions described by the authors are hyperostosis frontalis interna, osteoma, Paget's disease (osteitis deformans), meningioma, myeloma, xanthomatosis and others. These conditions are discussed briefly and illustrated.

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26. Anthony, D. H.: The Evaluation of X-Ray Diagnosis in Ophthalmology, Rhinology, and Otolaryngology, J. Tennessee M. A. **41**:45 (Feb.) 1948.

27. McGovern, F. H.; Fitzhugh, G., and Cooper, G.: Skull Roentgenograms of Interest to the Otolaryngologist, Ann. Otol., Rhin. & Laryng. **57**:387 (June) 1948.

Turesson [28] describes the technics employed for demonstrating fluid in the frontal and sphenoid sinuses. A fluid level seen in the posteroanterior projection with the patient's head erect can be corroborated by tilting the patient's head to one side and shooting the ray exactly the same as in the posteroanterior projection. The fluid level will have changed in relation to the sinus walls, while maintaining the horizontal. For roentgenography of the sphenoid sinuses a shelf is used which is screwed onto the Lysholm table, so that both full axial and horizontal projections may be taken with a horizontal ray, the patient remaining in a sitting posture. This is much more comfortable for the patient and provides two pictures showing changes in the fluid level. The article is well illustrated.

Filippi and Finocchi [29] discuss the advantages of tomography (planography) in transverse axial projection over conventional roentgenography of the sinuses. They claim that the partitions within the ethmoid cells, the maxillary extensions and the bony thickness of the frontal walls, as well as the accurate delineation of tumors, are much more clearly demonstrated by this method. At least they consider the procedure a valuable adjunct to the ordinary method producing flat pictures.

Grain and Bouche [30] report to the French Society of Electroradiology on their observations of roentgenologic procedures at the Massachusetts General Hospital, Boston. They describe the projections employed for the various sinuses, calling attention to the particular advantages of each. These include (1) the ventral decubitus for the lateral projection to show the thickness of the frontal sinus walls, depth of the sinus, degree of pneumatization and transparency of the ethmoid cells; (2) nose-chin with open mouth to show posterior ethmoid cells and sphenoid sinuses; (3) Hirtz position to show posterior ethmoid cells; (4) upright posteroanterior to show the ethmoid labyrinth; (5) upright nose-chin with open mouth to show fluid levels. The authors present several roentgenograms with comments on differentiation among fluid levels, cysts and diffuse edema due to allergy.

SINUSITIS IN CHILDREN

Archer [31] follows a procedure in the treatment of subacute maxillary sinusitis similar to that of Alden, although he states he was unaware of

28. Turesson, D.: The Roentgen Diagnosis of Fluid in the Frontal and Sphenoidal Sinuses, Acta radiol. **29**:215, 1948.

29. Filippi, P., and Finocchi, G.: La stratigrafia assiale trasversa in otorinolaringoiatria, Inform. Med., Genova **2**:139 (April) 1948.

30. Grain and Bouche: Présentation de clichés intéressant les sinus de la face et les rochers, J. de radiol. et d'électrol. **29**:138, 1948.

31. Archer, G. E.: The Treatment of Subacute Maxillary Sinusitis Especially in Children, Proc. Roy. Soc. Med. **50**:854 (Dec.) 1947.

Alden's work until long after. It consists of the introduction of an indwelling catheter through an intranasal antrostomy opening, with irrigations two or three times daily for seven to ten days. For small children a general anesthetic is employed; all others are treated under local anesthesia. An analysis of 100 patients showed 61 cured, 28 improved, 6 unchanged and 5 for whom results were not recorded. Conditions complicated by deviated septum or nasal polypi yielded rapidly after correction of these abnormalities. Archer's criteria for determining results were disappearance of subjective symptoms, subsidence of discharge and clearing in the roentgenograms. Removal of adenoids when present was also considered essential.

In the discussion that followed this presentation, Crooks stated that he had treated 570 children in the past year with roentgenologic evidence of maxillary sinusitis, 230 of whom showed clearing at the first irrigation. Of the 340 remaining, all but 9 recovered after repeated irrigations. When this method fails, and particularly in bronchiectatic children, he resorts to the Caldwell-Luc procedure, having performed it in 61 children, of whom 34 had bronchiectasis.

A follow-up study [32] of 1,779 children who had had their tonsils and adenoids removed revealed the fact that unsatisfactory results were in most cases due to the presence of chronic maxillary sinusitis. This accounted for the persistence of such symptoms as mouth breathing, snoring, enlargement of cervical nodes, pain in the ears, cough, nasal discharge, nightmares and recurring colds. A diagnosis of maxillary sinusitis was made for 290 children, or 16 per cent of the total series. Sinus irrigations and displacement therapy with weak penicillin solutions resulted in cure in 133 of them. In 128 children some symptoms still persisted. Patients whose conditions failed to improve after six weekly irrigations were subjected to bilateral antrostomy.

Laing,[33] in observations on 900 children treated by roentgen irradiation for subacute sinusitis, reports 71 per cent cured after one series of treatments. Nasal smears on all these children showed no evidence of allergy as a contributing factor. Roentgenograms showed varying degrees of cloudiness of the ethmoid cells and maxillary sinuses. For young children, all local nasal therapy was avoided, even the use of nose drops. If the nasal obstruction was severe, the initial dose of roentgen irradiation was very small (30 r).

The following technical factors were employed: 130 kilovolts (peak), 5 milliamperes, with 3 mm. of aluminum filtration, with a half-value layer of 4.1 mm. of aluminum and a distance of 30 cm. For the older

32. Walker, F. M.: Tonsillectomy and Adenoidectomy: Unsatisfactory Results Due to Chronic Maxillary Sinusitis, Brit. M. J. **2**:908 (Dec. 6) 1947.

33. Laing, D.: The Roentgen Treatment of Subacute Sinusitis in Children, Radiology **50**:52 (Jan.) 1948.

children the factors were 140 kilovolts (peak), 5 milliamperes, with 0.25 mm. of filtration with copper and 1 mm. with aluminum, with a half-value layer of 0.42 mm. of copper, and a distance of 30 cm. Three treatments were given over a period of eight days, and a total of 240 r was given to each area. If cough was present, roentgen treatment was directed at the roots of the lung, and when there was evidence of tubal involvement these areas were also irradiated through a 7 by 7 cm. port in a large protecting lead shield. About three fourths of all the children evidenced some signs of reaction after the first treatment, such as stuffiness of the nose and increased cough. The temperature was elevated in only 5 per cent of the patients.

The author claims that the therapy is most effective if applied after the first week of an attack of sinusitis at a time persisting symptoms may lead to chronicity. A second series of treatments was given to those whose disorders failed to respond to the first and resulted favorably in more than half. In the children whose condition was uninfluenced by the therapy, the author feels that underlying undiagnosed allergy may have been the factor.

Burgess [34] mentions nasal discharge and obstruction as outstanding symptoms of sinusitis in children. The discharge is thick and tenacious and may be seen by rhinoscopy, as well as in the pharynx. Cough and low grade fever are also frequently observed. The author has noted a heavy musty odor about these patients. Good results were obtained in most of them by adequate shrinkage followed by displacement therapy. Surgical treatment is seldom indicated in uncomplicated infections.

Fabricant and Perlstein [35] report the p_H of nasal secretion in 44 infants and children to range from 5 to 6.7, which is comparable to the findings in adults (p_H 5.5 to 6.5). In a group of 17 children with acute rhinitis the p_H was 7 to 8 and in a group with subacute rhinitis 6.7 to 7.2. In those with allergic sinusitis the readings were from 7.2 to 7.3. These figures parallel the observations in adults with similar conditions and, therefore, point to the necessity of using only such nasal medication as will reduce the p_H to the normal degree of acidity.

Brown [36] classifies chronic and recurring cough in children under the following heads: recurring bronchitis, recurring asthmatic bronchitis, recurring asthma, chronic bronchitis, bronchiectasis and miscellaneous coughs. These are separately analyzed and found to be related to

34. Burgess, T. S.: Paranasal Sinus Infection in Infants and Children, J. M. A. Georgia **37**:44 (Feb.) 1948.

35. Fabricant, N. D., and Perlstein, M. A.: Hydrogen Ion Concentration of Nasal Secretion in Situ in Infants and Children, Arch. Otolaryng. **47**:765 (May) 1948.

36. Brown, E. E.: Recurring and Chronic Cough in Children: Role of Sinusitis, Northwest Med. **47**:435 (June) 1948.

chronic sinusitis, except for atopic asthma due to ingested foods and inhaled pollens and epidermal allergens and for the chronic coughs caused by inhalation of a foreign body or a mediastinal tumor. Diagnosis of the chronic sinusitis in other cases is made on the basis of such symptoms as frequent colds, nosebleed, snoring at night, aprosexia, anorexia, fetor oris and puffy lids. Roentgenographic appearance cannot always be depended on. The treatment recommended embraces the application of ephedrine nose drops and of hot wet towels to the face, the avoidance of chilling and use of vaccines and a cough mixture comprising ephedrine, camphorated tincture of opium, glycerin and water.

György [37] cites 22 cases of sinusitis in infants. Fourteen patients were observed in the prepenicillin days; 8 died. All the subsequent 8 infants treated with penicillin recovered. Six of these underwent surgical therapy of the sinuses, in 2 of whom merely incision and drainage of an externally pointing abscess was done. The author discusses the pathogenesis and concludes that osteomyelitis of the superior maxilla is the result of a spread via the dense capillary network of the bone, the maxillary sinus itself becoming involved secondarily. However, when the infection is limited to the antrum, it generally gains access through continuity of the mucosa.

Gundrum [38] reviews the etiologic factors in sinusitis in children, and the important diagnostic procedures, and presents an evaluation of the role of allergy. Differential diagnosis on this point is stressed, with emphasis on methods of obtaining secretion for cytologic study. Discussing the therapy of acute sinusitis, Gundrum mentions the generally accepted procedures, such as rest, small doses of atropine and calcium compounds, penicillin injections and aerosol treatment. He advises nose drops of a low p_H instilled in the lateral head-low position and cautions against the use of mild silver protein (argyrol) and oily drops. Irrigations are not practiced until all the acute symptoms have subsided. Patients with chronic disorders require a thorough allergic work-up with appropriate therapy. In addition, it may be necessary to irrigate the antrum either directly or via displacement. Regulation of the diet and correction of endocrine disorders play an important role in complete cure of the patient's condition.

INTRACRANIAL COMPLICATIONS

Emhart [39] states that the otorhinolaryngologist should treat all complications arising from infections of the ears and sinuses, because he is

37. György, E.: Treatment of Sinusitis in Children, Paediat. danub. **3**:97 (Feb.) 1948.

38. Gundrum, L. K.: Sinusitis in Children, Arch. Pediat. **65**:293 (June) 1948.

39. Emhart, O.: Complicaciones intracraneanas otorrinógenas, Rev. otorrinolaring. **7**:138 (Dec.) 1947.

in a position better to evaluate the relationship of the focus to the complicating extension and can care for both simultaneously. He presents with considerable detail 8 cases of lateral sinus thrombosis, 2 cases of longitudinal sinus thrombosis, 5 cases of meningitis, 16 cases of cerebral abscess and 11 cases of cerebellar abscess. Attention is directed particularly to the early symptoms, it being possible in many cases to determine the diagnosis even before the results of laboratory tests are available.

In 7 of the 8 cases of lateral sinus thrombosis, chills and fever were early and predominating symptoms. Headache and vomiting noted in 3 cases were attributed to involvement of the external meninges and extradural abscess. Periphlebitis was present in 5 cases and thrombophlebitis in 3. The jugular vein was not ligated in any of the cases, the sinus being isolated by packs, with exploratory puncture to determine its patency and wide incision if pus or thrombus was encountered. Sulfonamide and penicillin therapy was actively employed. There was 1 case of metastasis to the lungs which yielded to penicillin treatment. The diagnosis of involvement of the longitudinal sinus was not so easy. One case was not accurately diagnosed until autopsy, the disease having been taken to be tuberculous meningitis which was treated with streptomycin. In the second case suspicion was directed to the sinuses despite the absence of symptoms, and they were widely opened, revealing a focus which was treated. The patient recovered. A variety of cranial symptoms was noted. Meningeal signs were indefinite, with the spinal fluid findings normal in one case and suggestive of tuberculosis in the other. Involvement of the cortex was indicated by the loss of lower limb reflexes and an ascending hemiplegia. More characteristic were the external manifestations of obstruction to circulation, such as edema and swelling of the frontoparietal veins and edema over the frontal bones, which at first suggested osteomyelitis. The cases of meningitis were all due to otitic foci. Of the 16 cases of cerebral abscess, 14 were in the temporal lobe and 2 of traumatic origin were in the frontal lobe. Despite drainage and use of antibiotics, the mortality was 45 per cent. The 11 cases of cerebellar abscess were all due to an exacerbation of chronic otitis media.

According to Schubert,[40] experience with penicillin therapy in Germany has been limited by the cost and the comparative scarcity of the drug. Of necessity, therefore, sulfonamides were extensively employed from 1939 to 1943 with excellent results. However, he has noted that in the succeeding years the drugs were less effective in the same types of infections, a change he attributes to the generally impoverished state

40. Schubert, K.: Die Therapie der oto- und rhinogenen Komplikationen insbesondere mit Penicillin, Ztschr. f. Laryng., Rhin., Otol. [**27**]:79, 1948.

of health of the entire community toward the end of the war and immediately thereafter. He also presents evidence to show how certain strains of bacteria have become resistant. In these cases the author has observed that a combination of sulfonamides with penicillin yields ideal results, and he presents several histories of cases of intracranial complications illustrating this point. In one case of extensive pansinusitis with phlegmon of the orbit, necessitating complete exenteration and wide exposure, the initial improvement with penicillin therapy was not sustained until a sulfonamide was given in combination. The explanation lies in the fact that in a mixed infection it is always possible to have some strains which are penicillin resistant. In this particular case the hemolytic streptococci yielded to the penicillin, whereas the Escherichia coli, the gram-positive *Lanzettkokken* and gram-negative bacilli were resistant. Comparison of dosages of penicillin brought out the fact that the amounts employed in this country are much larger than those used on the Continent.

Apparently, however, the point of the whole article is the importance of proper study of the bacterial flora in each case and the application of such antibiotics as are known to be effective for the particular strains in evidence.

Larroudé [41] reports 2 cases of osteomyelitis and abscess of the frontal lobe complicating acute frontal sinusitis in girls, aged 13 and 14, both of whom recovered. He comments on the difficulty in diagnosing the lesion of the brain before symptoms of cerebral pressure appear, since headache, fever and vomiting are characteristic of all three conditions. There is also the fact that the frontal lobe is a silent area. In both his patients however, fundus changes appeared early, as did paresis of the abducens, symptoms which pointed to intracranial involvement. While the favorable outcome may have been due in large measure to the liberal use of antibiotics, careful study of the case histories shows conservative therapy in the beginning, limited to simple drainage of the apparent subperiosteal abscess. The author believes that when an extradural abscess is uncovered in these cases, with granulations on the dura, one may assume that the suppuration has extended to the endocranium and is, therefore, justified in exploring for it.

Laszlo [42] reports a case of fatal air embolism six days following antral puncture and lavage. The patient was a 46 year old man. An attempt to pass a catheter into the natural opening was unsuccessful. After this, a Douglass cannula was introduced by puncture through the

41. Larroudé, C.: Dois casos curados de sinusite frontal aguda complicada de osteomielite do frontal e abcesso do cerebro, Rev. españ. oto-neuro-oftal. y neurocir. **4**:313 (Sept.-Oct.) 1947.

42. Laszlo, A. F.: Fatal Air Embolism During Antral Irrigation, Ann. Otol., Rhin. & Laryng. **58**:479 (June) 1949.

inferior meatus and the irrigation carried out without difficulty. Air was introduced after the irrigation and immediately afterward the patient "complained of feeling funny, became rigid, was seized with convulsions, fell to the floor and ceased breathing." All efforts at resuscitation failed. Autopsy revealed the heart and inferior vena cava distended with air. Examination of the sinus failed to show any damage to the mucosa other than the puncture. The author emphasizes the importance of correct technic in performing a puncture or washing through the natural opening. The end of the instrument should be felt to be freely movable to insure its being within the cavity. He presents arguments as to whether postirrigation insufflation of air is necessary.

Buzoianu, Popvici and Pop [43] report a case of meningitis in a man 63 years of age with recovery attributed to the administration of large doses of sulfonamides. The infection came about after an operation on the frontal sinus performed for the closure of a fronto-orbital fistula which resulted after enucleation for an orbital neoplasm. During the course of the operation, in which an unusually dense frontal cortex was removed, the cribriform plate was broken, resulting in laceration of the meninges and discharge of cerebrospinal fluid. Meningeal symptoms appeared the next day, despite early treatment with sulfonamides. However, the therapy was stepped up, the patient receiving a total of nearly 50 Gm. of sulfapyridine in six days. The patient was afebrile by the tenth day and recovered completely. The only local treatment mentioned was the primary application of a 5 per cent phenol solution on gauze and provision for drainage via the frontal wound, the orbit and the nasal cavity.

Schürmann's case [44] was interesting. The patient was admitted to the hospital in a comatose state with a history of severe headache of six days' duration following a cold. Purulent secretion was noted in the middle meatus, and the frontal, ethmoid and sphenoid sinuses appeared cloudy in the roentgenograms. Lumbar and occipital puncture revealed bloody fluid with 10,000 leukocytes per cubic centimeter. The frontal and sphenoid sinuses were opened, exhibiting pronounced inflammation without suppuration. The bony walls were intact. The infection was a fulminating one and the patient died within twenty-four hours. Autopsy disclosed a pituitary tumor grossly infected, with necrosis in certain areas. Microscopic examination of the tissues showed that apparently the infection had passed from the sphenoid sinus to the pituitary via the communicating vessels and that hemorrhage into the posterior

43. Buzoianu, G.; Popvici, G., and Pop, V.: Meningitis of Nasal Origin in an Old Man Cured by Intensive Sulfonamide Therapy, Rev. sc. oto-rhino-laryng. **11**:68 (Jan.-April) 1947.

44. Schürmann, F.: Endocranielle otogene oder rhinogene Komplikationen und bluthaltiger Liquor, Ztschr. f. Laryng., Rhin., Otol. [**27**]:224 (Aug.-Sept.) 1948.

fossa had resulted from destruction of vessels in the tumor. The author states that similar cases had previously been reported by Kraus and Hajek. It appears that, in Schürmann's case as in one reported by Alexander, there had been no previous indication of the existence of the pituitary neoplasm.

In the case reported by Buzoianu, Popovici and Moraru [45] a woman, aged 22, showed meningeal symptoms and swelling of the lids following digital trauma to a furuncle in the frontal region. Spinal puncture revealed purulent fluid but no bacteria. Since the initiation of sulfonamide therapy failed to halt the process, a mastoid operation was done, revealing normal bony structure after the uncovering of a thrombosed lateral sinus. The jugular vein was ligated and penicillin therapy begun. A total of 2,000,000 units and approximately 200 tablets of sulfonamide were given. The ocular symptoms receded; pain lessened with the improvement in the meningeal condition, and recovery was complete in a little over ten days. The absence of involvement of the mastoid cells despite the thrombosis of the lateral sinus is explained on the basis of an infection proceeding from within outward, or rather from the veins in the frontal area by way of their communication with the endocranial venous channels.

Fiori-Ratti [46] reports a case of multiple abscesses of the frontal lobe on the side opposite a sinus infection which developed several months after surgical treatment for the relief of an acute condition. He comments on the difficulties in diagnosis, which was finally achieved with the help of ventriculograms. Predominant symptoms were the psychic disturbances, as well as the cerebellar dysfunction, occasionally noted in disease of the frontal lobes. He seems to think the cerebral complication was the end result of septicemia, although the observations at operation disclosed extensive osteomyelitis. [It should be noted that the initial surgical procedure performed when the illness was still very acute consisted of opening of the sinus and enlargement of the nasofrontal duct. S. S.]

Baker, Simonton and Fisher [47] report a case of pneumocephalus with hemiplegia and choked disks due to an osteoma but without evidence of rhinorrhea or meningitis. The patient was a man 26 years old whose illness began with afternoon headaches accompanied with nausea and

45. Buzoianu, G.; Popovici, G., and Moraru, G.: Thrombophlebitis of the Cavernous Sinuses Accompanied by Thrombophlebitis of the Right Lateral Sinus, and Puriform Meningitis, Cured by a Triple Action: Penicillin, Sulfonamide, Surgical Treatment, Rev. sc. oto-rhino-laryng. **11**:3 (Jan.-April) 1947.

46. Fiori-Ratti, L.: Ascessi encefalici frontali multipli da sinusite frontale controlaterale, Policlinico (sez. prat.) **55**:753 (June 21) 1948.

47. Baker, G. S.; Simonton, K. M., and Fisher, R. G.: Pneumocephalus with Hemiplegia Secondary to Osteoma of Frontal Sinus: Report of Case, Proc. Staff Meet., Mayo Clin. **23**:528 (Nov. 10) 1948.

vomiting. Stooping, sneezing and blowing increased the headache, which was felt at the base of the skull with extension to the vertex and frontal region. There was some weakness in the right arm and leg. Roentgenograms showed a large collection of air in the right fronto-parietal region of the brain; also an opacity was noted in the right frontal sinus which appeared to be an osteoma. The patient was given sulfadiazine and crystalline procaine penicillin G (duracillin®) and then subjected to bilateral frontal craniotomy, with removal of an osteoma 4 cm. in diameter and "exploration of a cavity filled with air which presumably had been an old brain abscess." The patient recovered.

Grimaud, Neimann and Chalnot [48] report an interesting case of cerebral edema complicating acute frontoethmoid sinusitis in a child 9 years of age. The initial edema of the lids and forehead and the severe headache seemed to subside several days after the onset of the infection, when the child was suddenly seized with convulsions and went into a coma. Clonic twitching of the right half of the face and seizures involving the arm and leg on the same side were noted. However, there was no Babinski sign and little stiffness of the neck. Spinal puncture revealed clear fluid under great pressure. Thirty cubic centimeters was evacuated and 20 cc. of serum containing 20,000 units of penicillin injected. The frontal tumefaction was incised, liberating about 1 cc. of pus. The bone was not involved. Nevertheless, the sinus was opened and noted to be full of pus and granulations. The posterior wall was removed and the meninges appeared normal. Aspiration in various directions failed to uncover any cerebral abscess. The recovery was prompt. The convulsions ceased and the child regained consciousness and rapidly returned to normal. The authors discuss the differential diagnosis, demonstrating how they excluded meningitis, thrombosis of the cavernous and longitudinal sinuses, and conclude that the only possible diagnosis was cerebral edema.

Dylewski [49] reports 2 cases of purulent frontal sinusitis complicated by epileptiform seizures with no previous history of epilepsy. The first case was of acute sinusitis in a patient 16 years of age. Headache, fever and swelling over the brow were followed by unconsciousness and meningeal symptoms. The frontal sinus was opened, releasing foul pus, softened bone and degenerated mucosa. Consciousness returned the next day but the patient had aphasia. On the fifth day typical jacksonian seizures occurred, with loss of consciousness. The spinal fluid

48. Grimaud, Neimann and Chalnot: Œdème cérébral grave au cours d'une sinusite fronto-ethmoïdale chez une enfant de neuf ans, Ann. d'oto-laryng. **65**:180 (March-April) 1948.

49. Dylewski, B.: Purulent Frontal Sinusitis Complicated by Epilepsy: Two Cases, Otolaryng. polska **2**:70, 1948.

was under normal pressure, with a slightly positive reaction for albumin, a doubtful Pandy reaction and no cells. The attacks continued for several days and finally disappeared with restoration of speech. The author concludes that the symptoms were caused by cerebral edema due to stasis.

The second case was that of a patient 26 years old with a history of headache for four weeks following an acute infection of the upper respiratory tract. Local swelling and epileptiform attacks appeared, with fever and meningeal symptoms. The spinal fluid gave a positive reaction for albumin and a positive Pandy reaction and showed 24 cells per cubic millimeter but no bacteria. Both frontal sinuses were opened, revealing pus, diseased bone and degenerated mucosa. The patient's condition improved for six days, after which there was a recurrence of the headache and septic temperatures. The swelling of the forehead reappeared, extending up beyond the hair line. Temporary relief followed administration of sulfonamides and penicillin, but on the twenty-second day the symptoms again appeared, at which time the wound was reopened and extended toward the vertex. A softened area of bone was encountered above the limits of the sinus, which on removal uncovered a large extradural abscess. Recovery was complete.

Ireland [50] discusses briefly the pathways of infection from the ear and the sinuses to the endocranium and emphasizes the importance of close collaboration between the otolaryngologist and the neurologist.

OCULAR AND ORBITAL COMPLICATIONS

D'Aiutolo and Longhena [51] claim that the introduction of radiopaque fluids into the posterior sinuses by the Proetz displacement technic has a beneficial effect in cases of optic neuritis. This was particularly evident in those cases in which clinical symptoms of sinusitis were present. When such symptoms were absent, the favorable results were attributed to action on the nasal and paranasal reflexes or to the liberation of histamine-like substances. The clinical histories of 20 cases are given in brief, showing the improvement in vision and the visual fields. Seven cases of optic neuritis were definitely ascribed to a sinus infection and improvement was noted on treatment in each case. In 2 of these the improvement was only transitory. Surgical intervention, however, brought about a more rapid return to normal. Six cases were diagnosed

50. Ireland, P. E.: Intracranial Complications of Ear and Sinus Disease, Bull. Acad. Med., Toronto **21**:92 (Feb.) 1948.

51. d'Aiutolo, F., and Longhena, L.: Influenza dell'introduzione di liquidi radiopachi nelle cavità paranasali con il metodo dello spostamento di Proetz in alcuni casi di lesioni del nervo ottico, Riv. oto-neuro-oftal. **22**:335 (Sept.-Dec.) 1947.

as arachnoiditis of the chiasm. Two of them showed definite improvement and a third case yielded only moderately to treatment. Two were unaffected. Symptoms of sinusitis were present in only 3 cases of this group.

On the basis of 12 cases of orbital complications of sinus disease which are presented in abstract, Escher [52] discusses in detail the anatomic and physiologic characteristics, pathogenesis and therapy. His series included thrombosis of the cavernous sinus, osteomyelitis, meningitis and brain abscess. Roentgenograms and photographs of patients illuminate the article. The author points out the importance of avoiding intranasal surgical procedures in these cases. Antibiotic therapy is relied on in mild conditions, with puncture and irrigation of the maxillary sinus. If symptoms indicate suppuration in the orbit, external surgical treatment is necessary but should be limited to drainage only, unless there are signs of extension into the bony spaces or intracranially.

Among 12 of the cases in which operation was done pus was observed in 9, while in the other 3 only extensive edema with hemorrhagic areas was uncovered. The article is well written with just enough detail to make it impressive.

Gaini [53] describes three stages in the development of orbital infection due to sinusitis. The first is simple cellulitis without suppuration, characterized by edema of the lids, some conjunctivitis and moderate pain and fever, which should yield to antibiotic therapy if adequate intranasal drainage is effected. In the second, or more advanced, stage the signs of phlegmon are more definite but the general condition of the patient is good. Here antibiotic therapy is indicated. Surgical treatment will depend on the degree of response to the drugs and should be adequate but circumspect. In the third stage the ocular signs are pronounced and exophthalmos, fixation and loss of corneal reflex with fixation of the pupil are accompanied with symptoms of sepsis. These cases require wide exposure if permanent damage to the eye is to be avoided. In the 3 cases of orbital infection cited by the author, one was discovered to be due to syphilis and responded promptly to antisyphilitic therapy. In the others, severe reaction followed early primary intranasal surgical therapy, which necessitated a second external radical procedure. [It appears from the history that the early intervention was ill advised and was contrary to the principles generally accepted with regard to the avoidance of intranasal surgical procedures in the acute stages. S. S.]

52. Escher, F.: Die Komplikationen der Nasennebenhöhleneiterungen und ihre Therapie, Praxis **36**:829 (Dec. 11) 1947.

53. Gaini, G.: Nuovi aspetti clinici delle infezioni del cellulare orbitario di origine sinusale in rapporto con la terapia sulfamidica e penicillinica, Arch. ital. di otol. **58**:248 (Dec.) 1947.

Barnett and Ford [54] stress the importance of adequate resection of the nasal septum in order to provide free ventilation to the sinuses and assist in drainage. Too often the surgeon fails to remove a high deviation or one that extends far back. The authors also advise reduction of hypertrophied turbinates by submucous crushing of the bone. Certain ocular disturbances due to sinus infection are discussed and case histories cited. These included cases of ptosis, episcleritis, keratitis, iritis, choroiditis and retrobulbar neuritis, in which an infected sinus was observed to be the cause. These ocular manifestations occur most frequently when the sinusitis is acute, drainage being obstructed through either swelling of the tissues blocking the ostium or inspissated secretion, or both. In the cases cited, the ocular symptoms disappeared when the sinus was properly drained. The intimate association of ocular diseases with nasal lesions makes it imperative in all inflammatory processes of the eye to seek diligently for possible foci in the nasal sinuses.

Wilkinson [55] states that sinus disease is responsible for 70 per cent of the cases of orbital cellulitis and for 5 per cent of the cases of uveitis. There is no general agreement concerning the role it plays in retrobulbar neuritis. According to the author, the consensus is that not more than 3 per cent of these cases are due to sinus infection. These, along with the cases of malignant growths and cysts, are of interest to both the ophthalmologist and the rhinologist, as well as the roentgenologist, and, therefore, call for closer cooperation among these specialists.

Latorre [56] had a patient with acute ethmoid sinusitis with abscess formation at the external angle, which caused a transitory astigmatism of 3.5 diopters. The condition cleared after the abscess was drained and the ethmoid cells were cleaned out. The author claims that the astigmatism was due to pressure on the bulb causing distortion, and he points to the differential diagnosis from optic neuritis due to the infection, which might have been considered were it not for the careful refraction that was done in addition to the usual fundus examination.

Goodyear [57] lists a number of ophthalmic conditions caused by sinusitis, including retrobulbar neuritis, edema of the lids and intracranial infections, orbital abscess, hematoma and displacement of the

54. Barnett, H. N., and Ford, R.: Ophthalmic and Other Affections Predisposed to by Deformities and Caused by Sepsis of the Nose, M. Press **110**:239 (March 17) 1948.

55. Wilkinson, R. W.: Ocular Effects of Nasal Sinus Disease, Eye, Ear, Nose & Throat Monthly **27**:560 (Dec.) 1948.

56. Latorre, S.: Fuerte astigmatismo corneal transitorio en un caso de sinusitis etmoidal, Arch. Soc. oftal. hispano-am. **7**:1009 (Oct.) 1947.

57. Goodyear, H. M.: Ophthalmic Conditions Referable to Diseases of the Paranasal Sinuses, Arch. Otolaryng. **48**:202 (Aug.) 1948.

bulb due to tumors, and offers pertinent comments on diagnosis and therapy. He describes an adaptation of a nail drill found useful in opening the frontal sinus for drainage in acute cases. He calls attention to the advantages of the use of contrast mediums in diagnosis.

Cordato de Noronha [58] reports an usual case of exophthalmos and blindness in one eye due to retrobulbar hemorrhage and hemorrhage in the frontal sinus in a man 30 years of age who was syphilitic and a sufferer from chronic schistosomiasis. The other eye remained normal. Considerable improvement followed antisyphilitic therapy. The symptoms came on within five days, with redness of the bulb, increasing exophthalmos and diminishing vision to total blindness. Subjectively a feeling of pressure in the orbit and radiating pains were present. Examination revealed all the signs of retrobulbar pressure, roentgenologic evidence of a dark frontal sinus, retinal hemorrhages and thromboses and absence of any indications of infection. There was a history of his having contracted schistosomiasis at the age of 10; the Wassermann reaction was positive and spores of Schistosoma haematobium were recovered in the urine. There was no history of recent trauma. With antisyphilitic therapy the hematoma was absorbed and the frontal sinus showed up clear in the roentgenograms.

Tétu and Marin [59] reported 5 cases of orbital involvement and 4 of endocranial complication among a series of 170 cases of sinusitis. These were 3 cases of orbital abscess, 1 of frontal abscess with fistula and 1 of retrobulbar cellulitis with osteitis (due to pansinusitis of dental origin), 1 of meningitis (post-traumatic frontal osteitis), 1 of meningoencephalitis (of frontal sinus origin) and 2 of brain abscesses (of frontoethmoid and frontal sinus origin). Four cases are cited. The authors discuss the various pathways of infection in general and as applied to their own cases in particular. They emphasize the importance of thrombosis of communicating veins in these conditions and the necessity for wide excision of all diseased bone. Treatment embraces recognition of the pathogenesis, adequate drainage of the original foci, resection of all diseased tissues and the proper use of antibiotics.

OTHER COMPLICATIONS

Bouchet [60] calls attention to certain neuromuscular syndromes in the cervical, dorsal and, even, lumbar areas which are due to chronic infection of the sinuses. The symptoms are cervical rigidity, local

58. Cordato de Noronha, R.: Exophthalmie par hémorragie du sinus frontal et hématome rétro-bulbaire, Bull. Soc. d' opht. de Paris, June-Oct. 1947, p. 409.

59. Tétu, L., and Marin, A.: Complications orbito-crâniennes des sinusites de la face, Ann. d'oto-laryng. **65**:499 (Aug.-Sept.) 1948.

60. Bouchet, M.: Sinusites latentes et complicationes névritiques, Ann. d'oto-laryng. **64**:629 (Nov.-Dec.) 1947.

tenderness and enlargement of the cervical and submaxillary glands. Examination usually reveals redness and swelling of the lateral pharyngeal bands and involvement of one or more of the sinuses.

He is disinclined to attribute the process to a hematogenous spread of the infection but feels that passage through the lymphatics provides the true explanation. Infected secretion from the nose and sinuses drains into the prevertebral and retropharyngeal nodes and may extend retrograde via lymph channels to the muscles and even to the base of the brain. In this manner one can readily explain cervical rigidity, hypesthesia along the distribution of the brachial plexus, clonic movements of the upper extremities and meningeal symptoms. Drainage in the normal direction accounts for extension of adenitis into the submaxillary and cervical chain. The author cites 4 cases of this type in which proper attention to the sinuses resulted in prompt relief. He cites several experiments by Monnier-Vinard on the dispersion of india ink injected in the subarachnoid spaces, reaching the perimeningeal and perivertebral spaces and finally entering the prevertebral lymph nodes.

Réthi [61] draws a distinction between trigeminal neuralgia and neuritis of certain branches of the nerve. The former comes on suddenly in an apparently healthy person, is severe for a while and subsides quickly, leaving no after-effects. Neuritis, on the other hand, never attains the degree of severity of neuralgia and persists for longer intervals with varying degrees of intensity. The patient may be cured of the latter but is always subject to sudden recurrences of the former; also the local tenderness is more persistent in neuritis than in neuralgia. Certain pathologic states in the sinus may cause true neuritis and, when the diagnosis is well defined, relief may be had through injecting into the nerve canal 0.5 cc. of a 3 per cent solution of hydrogen peroxide. The author has employed this procedure successfully in treatment of disease of the frontal and maxillary sinuses, injecting either the supraorbital or the infraorbital nerve, as the case may be.

Adams and Ficarra [62] analyze the results in 50 consecutive patients with bronchiectasis treated surgically at the Lahey Clinic, Boston. Although the subject of sinusitis as an etiologic factor is not discussed, it is interesting to note that this condition was present in 20 of the 50 patients.

Allende and Langer [63] report 2 cases of Kartagener's triad (situs inversus, bronchiectasis and sinusitis), with roentgenograms and photo-

61. Réthi, A.: Névrites trigémellaires d'origine rhinogène et leur traitement, Ann. d'oto-laryng. **64**:431 (Aug.-Sept.) 1947.

62. Adams, R., and Ficarra, B. J.: Appraisal of Surgery in the Treatment of Bronchiectasis, J. A. M. A. **134**:240 (May 17) 1947.

63. Allende, J. M., and Langer, L.: Transposición de visceras, bronquiectasia y sinusitis: Síndrome de Kartagener, Cirugía **1**:176 (Oct.-Nov.) 1947.

micrographs of sections from the extirpated lobes. One case was that of a boy of 17 and the other that of a girl of 14. The lower lobe of the left lung was removed from the first patient and a segment of the upper lobe from the second. The clinical histories are well written, and, in addition, the authors discuss various theories that have been advanced to explain the syndrome. The incidence of situs inversus in connection with bronchiectasis and sinusitis seems to be increasing as statistics accumulate, and the authors feel that, as a result of these studies, one cannot escape the conclusion that there is a hereditary and congenital basis for the development of bronchiectasis, particularly in young persons, and that the sinusitis which is usually present may develop *pari passu* with the pulmonary condition or appear even at a later date.

OSTEOMYELITIS

Maxwell[64] observes a difference in type of complications of frontal sinusitis according to age groups. In children with acute fulminating infections the spread is likely to be along venous channels and to result in extracranial complications, whereas in adults the complications are more frequently intracranial. Complications of chronic sinusitis usually result from an acute exacerbation and will be calvarial, at least in the beginning.

The symptoms suggesting a spread of the infection beyond the sinus are listed and typical symptom groups illustrated with case histories. Periorbital abscess, orbital cellulitis, cavernous sinus thrombosis, subperiosteal frontal abscess, osteomyelitis and dural abscess are all discussed from the point of view of diagnosis and therapy. The author goes into some detail in describing the surgical procedures on the frontal bone in osteomyelitis, advising exposure by the coronal incision with piecemeal removal of bone over a wide area. This provides better visualization of the extent of the invasion and facilitates adequate drainage into the nose.

The case recorded by Lewis[65] presents some interesting as well as puzzling data. The condition of a 20 year old man with a history of headaches for over a year became steadily worse in the last twelve days, and he was admitted to the hospital with left-sided hemiplegia and meningeal symptoms. The nasal findings indicated bilateral maxillary and frontal sinusitis. He was given penicillin intramuscularly and intrathecally, and within twenty-four hours a fluctuant swelling devel-

64. Maxwell, J. H.: Complications of Frontal Sinusitis, Tr. Indiana Acad. Ophth. & Otolaryng. **30**:68, 1947.

65. Lewis, R.: Osteomyelitis of the Skull Complicating Frontal Sinusitis, J. Laryng. & Otol. **62**:88 (Feb.) 1948.

oped over the left parietal area. There was evidence of bone absorption in this area. At operation pus was observed here, with some softened bone and an extradural abscess. No attempt was made to remove all the diseased bone. The frontal sinuses were opened and, although the dura was normal, the brain was probed on the assumption that an abscess of the frontal lobe was present. No pus was encountered. Penicillin was given and improvement was noted for four weeks. However, at this time swelling recurred in the parietal region and it was again explored, revealing more diseased bone and an extradural abscess. The spinal fluid pressure was high during most of this time, but no bacteria were recovered. Penicillin and sulfamethazine were again administered and improvement followed, until at the end of six weeks the patient had recovered except for some weakness in the leg. After four months recovery was complete.

The author attributed the appearance of osteomyelitis in the parietal bone to an extension by way of the veins. The causal organism was not determined but may have been anaerobic, since cultures were not made for such organisms. The diagnosis of left-sided hemiplegia when there was no lesion in the right hemisphere was explained on the basis of thrombosis of the superior longitudinal sinus and superficial cerebral veins.

Carnevalle-Ricci[66] presents the histories in detail of 5 persons with osteomyelitis of the frontal bone, each of whom recovered. In 2 the osteomyelitis was due to chronic frontal sinusitis, in 2 to acute infections and in 1 to probable trauma. He also enters into a full discussion of the diagnosis and therapy. He attributes his good results to the extensive removal of the diseased bone and adjacent tissues, although 3 of the patients had the benefit of penicillin therapy. It is interesting to note, however, that in some of the patients several operative procedures were undertaken before the process was halted. The histories indicate that the early operations were all inadequate, so that recurrence of symptoms necessitated further surgical intervention. [This demonstrates the importance of proper interpretation of the clinical findings, so that surgical treatment, when indicated, may be undertaken at the time the chances of reaching the limits of the process will be the most favorable. S. S.]

Commenting on the relative infrequency of acute frontal sinusitis, Johnson[67] states that a review of the files of the Los Angeles General Hospital over a ten year period revealed only 68 patients with this disease requiring hospitalization out of a total of 51,692 patients seen in

66. Carnevalle-Ricci, F.: Sull'osteomielite della teca cranica da sinusite frontale, Arch. ital. di otol. **58**:173 (Oct.) 1947.

67. Johnson, E. D.: Acute Frontal Sinusitis, Arch. Otolaryng. **47**:165 (Feb.) 1948.

the clinic. Osteomyelitis developed in only 6 of these. The cases of 2 patients are cited. One was a man 38 years of age, the other a girl 18 years old. The former died after four weeks, the autopsy showing an abscess of the frontal lobe and extension of the osteomyelitic process into the malar and temporal bones; although sulfonamides were administered, the focus was not attacked radically. The decision to exenterate the frontal bones was deferred until the patient's condition was so poor that the procedure was abandoned. In the second patient penicillin was given and the focus drained. Radical surgical therapy was advised but refused by the patient. Subsequently, when symptoms of a brain abscess appeared, the cranium was opened but no abscess was observed. Nevertheless, the paralysis disappeared and with heavy doses of penicillin the patient recovered.

After citing the work of Mosher and others on the diagnosis and therapy of osteomyelitis due to disease of the frontal sinus, Bleidt [68] concludes that early drainage and the combined administration of penicillin and sulfonamides are indicated in treatment of acute frontal sinusitis. Simple trephination through the floor should be employed if medical treatment and intranasal shrinkage fail to improve the condition. The incidence of osteomyelitis has definitely declined since the advent of antibiotics and the more general adoption of trephination.

The case reported by McKenzie [69] is interesting because it was seen in two distinct phases. In the first phase penicillin therapy probably saved the patient's life, and in the second it limited the spread of the osteomyelitis. On his admission, the 38 year old man was unconscious and had an elevated temperature, edema of the forehead and symptoms of acute frontal sinusitis. Smears yielded Staphylococcus pyogenes var. aureus. He was given 30,000 units of penicillin every four hours and the sinus drained simply through an external incision. This therapy, continued for ten days, resulted in apparent cure. He was discharged from the hospital despite the fact that he had fallen and bruised the forehead the previous day. He returned a month later with swelling of the forehead and roentgenologic evidence of osteomyelitis. An operation disclosed a wide area of diseased bone. A drainage tube was left in the cavity and penicillin solutions were instilled at intervals, while at the same time he was given 100,000 units every three hours intramuscularly. The tube was removed after three days and the administration of intramuscular injections of penicillin maintained for three weeks, to a total of 16,800,000 units.

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68. Bleidt, L. C.: Complications of Sinusitis, with Particular Emphasis on Osteomyelitis of the Frontal Bone, J. M. A. Alabama **17**:337 (April) 1948.

69. McKenzie, W.: A Case of Osteomyelitis of the Frontal Bone Treated by Penicillin, J. Laryng. & Otol. **62**:33 (Jan.) 1948.

Glass [70] credits penicillin therapy in no small measure for the recovery of his patient, who survived a stormy course lasting almost three months. The acute frontal sinusitis was complicated by convulsions, jacksonian attacks and transient hemiplegia which was attributed to vascular thrombosis in the motor area. Extensive surgical exploration uncovered a large extradural abscess and considerable diseased bone. A gutter was cut through the outer table at a safe distance beyond the diseased bone, which seemed to halt the process. At a later operation the diseased external table was removed, but the inner table was left alone, since it bled freely and appeared to be viable.

Mihaesco [71] reports a case of diffuse osteomyelitis due to a fronto-ethmoid infection in which treatment with penicillin was remarkably effective when first employed but failed to save the patient when he suffered a relapse and the drug was again administered. The infecting organism was Staph. pyogenes var. aureus. The author poses the question whether the failure was due to insufficient dosage of the antibiotic in the first place or to overmuch reliance on its efficacy. He believes that the penicillin should be combined with sulfonamides in treatment of such cases. [It should be noted that the dosage employed in the first instance was only 50,000 units per day, which is hardly adequate in the light of present day practice. S. S.]

Brunner [72] now classifies osteomyelitis according to etiologic factors and cites cases under five divisions, viz., osteomyelitis due to metatasis, osteomyelitis caused by trauma, osteomyelitis in chronic frontal sinusitis, osteomyelitis in acute frontal sinusitis and postoperative osteomyelitis. Osteomyelitis due to acute frontal sinusitis and that following surgical intervention have an unfavorable prognosis. In the traumatic type involvement of the sinus is secondary and may be delayed. The author reports antibiotic therapy of little value in the primary cases but useful in cases caused by acute frontal sinusitis as well as in those due to chronic infection.

MALIGNANT TUMORS

Crozet [73] analyzes a series of 155 cases of malignant growth of the nose and sinuses, discussing location, type of growth and age incidence. Among his conclusions are the following:

1. Primary malignant growth of the frontal and sphenoid sinuses is rare.

70. Glass, G.: Case of Osteomyelitis of Frontal Bone Following Acute Frontal Sinusitis, Treated by Surgery and Penicillin, J. Laryng. & Otol. **62**:310 (May) 1948.

71. Mihaesco, P.: Considérationes su l'action de la penicilline dans un cas d'osteite cranienne diffuse, Rev. sc. oto-rhino-laryng. **11**:52 (Jan.-April) 1947.

72. Brunner, H.: Osteomyelitis of the Face and Skull, Illinois M. J. **93**:213 (April) 1948.

73. Crozet, A.: Considerations statistiques sur les tumeurs malignes des fosses nasales et des cavites accessoires, Rev. de laryng. **69**:179 (March-April) 1948.

2. The two sides of the nose are affected in the same frequency.

3. Tumors of the ethmoid cells are usually cylindric epitheliomas, while those of the maxillary sinus are of squamous cell origin.

4. Malignant tumors of the nasal passages are rare before the age of 30.

5. Men are affected oftener than women.

The author has failed to observe a single case of malignant growth primary in the septum. Tumors of the maxillary sinuses are commoner than those of the nasal cavity proper. Most of the tumors observed involved the anterior portion of the nose.

Schall [74] reviews the methods of treatment in a series of 219 patients with malignant tumors of the nose and accessory sinuses seen at the Massachusetts Eye and Ear Infirmary from 1930 to 1945. Only 10 per cent of these could not be traced, but, of the remainder, 30 per cent survived five or more years. The greater number of these received surgical therapy primarily, whereas the minority were treated principally by irradiation, although in both groups the two procedures were employed. The author advises wide exposure in all operable conditions, with complete exenteration of the affected areas. In some patients the cutting current is used. The operation is followed by an adequate dose of radiation through the incorporation of radium capsules within the packing placed in the exenterated cavity. A total of 2,000 to 4,000 r is administered, depending on the sensitivity of the tumor and its extent. A study of this series shows that patients treated primarily by surgical methods supplemented with irradiation (either by radium or by high voltage roentgen rays) have a better than 2 to 1 chance of a five year survival than those treated primarily with irradiation. Several instructive cases are cited.

Godtfredsen [75] discusses the ophthalmologic symptoms produced by malignant tumors of the sinuses, based on personal observation in 14 cases. Statistics show that only one fifth of cancers of the sinuses penetrate into the orbit. Invasion of the orbit is characterized by early exophthalmos, with or without trigeminal neuralgia, displacement of the bulb depending on the direction of growth of the tumor, impaired ocular mobility, obliquity of the palpebral fissure, palpable tumor in many cases and impaired vision. The maxillary sinus is the primary site of the growth in most cases. Roentgenograms are of great assistance in delineating the extent of the growth. The author points out the fact that at times it is necessary to differentiate these tumors from orbital

74. Schall, L. A.: Malignant Tumors of the Nose and Nasal Accessory Sinuses, J. A. M. A. **137**:1273 (Aug. 7) 1948.

75. Godtfredsen, E.: Studies on Orbital Tumors: Exophthalmos Due to Malignant Tumors in the Paranasal Sinuses, Acta ophth. **25**:295, 1947.

involvement secondary to nasopharyngeal carcinoma. In the latter, orbital invasion comes late, is associated with more pronounced nasal symptoms and early nodular involvement and is more frequent in younger persons. In the author's experience malignant growths of the sinuses invading the orbit occur far oftener in older persons.

Szpunar [76] reviews a series of 51 patients with malignant tumor of the nasal sinuses treated at the Cracow University, Poland, from 1945 to 1948. The author is impressed with the results reported by New and Ohnrgren through surgical diathermy and irradiation but states that, owing to lack of adequate equipment, he was unable to apply this technic to his patients. These were treated with surgical excision followed by irradiation. Twenty-one patients were operated on via the Denker approach, 2 by lateral rhinotomy, 3 by the Killian route, 1 by the Preysing incision and 2 by atypical technics. Diathermy was employed to a limited extent after surgical treatment and before irradiation. In 4 patients metastatic nodes were excised and the area was irradiated. There was no primary mortality. The cases are too recent to permit long range evaluation. However, at the time of writing, 7 of the 27 patients operated on were free from evidence of disease one-half to one and one-half years later. In 8 patients local or nodular recurrences were noted; possibility of improvement in 5 patients was regarded as doubtful from the beginning (because of reoperation or incomplete removal), and 7 patients could not be traced. In those with inoperable tumors the author suggests use of nitrogen mustard (*tris* [β-chloroethyl] amine hydrochloride or methyl-*bis* [β-chloroethyl] amine hydrochloride) as a palliative.

McKenzie [77] briefly describes the symptoms of malignant growths of the sinuses and indicates the usual therapy. Usually a combination of surgical treatment, electrosurgical therapy and irradiation is necessary. Surgical treatment should be as radical as necessary without regard for the possible cosmetic deformities. These can always be corrected at a later date. The author recommends postoperative irradiation in all surgical cases.

Pietrantoni [78] states that a study of the zones of neuralgia and anesthesia about the face and mouth will often yield valuable data in the early diagnosis and localization of malignant tumors of the maxilla. Four cases are cited. The early association of anesthesia in the cheek with

76. Szpunar, J.: Treatment of Malignant Tumors of Nasal Sinuses, Acta otolaryng. **36**:317, 1948.

77. McKenzie, W. R.: Malignancy of the Nasal Sinuses, South. M. J. **41**:156 (Feb.) 1948.

78. Pietrantoni, L.: Zone nevralgiche e zone di anestesia della regione facciale e della cavità orale come sintomi precoci di alcune forme di tumori maligni delle cavità paranasali, Arch ital. di otol. **59**:105 (May) 1948.

anesthesia of the upper molars, when accompanied with radiopacity of the sinus, presents a syndrome that is characteristic of a malignant tumor arising from the posterior wall. The author admits that pain in the distribution of the trigeminal branches may be caused by various endocranial lesions, as well as by tumors in the nasopharynx, but points out the importance of always keeping in mind the possibility of a tumor of the maxillary sinus which proper roentgenologic examination should identify.

With reference to primary carcinoma of the ethmoid cells, pain and anesthesia are more likely to appear late than early, unless the nasal obstruction is severe. In such cases the zone of anesthesia involves the anterior third of the septal mucosa and the corresponding lateral wall of the nose. Tumors of the posterior ethmoid cells and sphenoid sinus are likely to involve branches of the sphenopalatine nerve, with pain and anesthesia of the palatal mucosa and posterior part of the nasal cavity.

The article has several diagrams showing areas on the face, on the palate and in the nasal cavity affected by involvement of various branches of the trigeminus.

Bozzi [79] had a patient with carcinoma involving the lower portion of the maxillary sinus, alveolus and palate, which was removed by resection of the superior maxilla. A number of slides is shown depicting various histologic aspects of the growth and considerable discussion is developed in regard to its true nature. It was diagnosed as an undifferentiated carcinoma simplex with certain features suggesting endothelioma. Growths of this type are not suitable for irradiation therapy, and, inasmuch as they grow slowly by erosion as well as infiltration of adjacent structures, they are more amenable to radical surgical removal. An interesting feature of this case was the excellent roentgenograms, including planigrams, which outlined the exact limits of the growth and proved helpful in planning the surgical procedure.

Arons and Oppenheim [80] report 2 cases of extensive carcinomas of the maxillary sinus which were treated with high voltage roentgen irradiation. The results were only palliative.

Noves Ubach [81] had 3 patients with malignant tumor of the maxillary sinus in one year, 2 of whom died within a few months. In these patients, malignant growth was not suspected until the sinus had been operated on for the relief of chronic sinusitis. In both patients, after the biopsy

79. Bozzi, E.: Carcinoma indifferenziato telangectasico del seno mascellare (diagnosi differenziale fra ca. simplex ed endotelioma), Arch ital. di otol. **59**:222 (Sept.) 1948.

80. Arons, I., and Oppenheim, A.: Carcinoma of the Maxillary Antrum, Harlem Hosp. Bull. **1**:65 (Sept.) 1948.

81. Noves Ubach, L.: Tumores malignos de seno maxilar, An. med., Barcelona **34**:316 (Aug.) 1947.

specimen revealed the true nature of the condition, radiation therapy was tried without effect. In the third patient, the orbit was exenterated and the superior maxilla resected. A good primary result was obtained, but there was no comment concerning the ultimate outcome. No mention was made of the use of surgical diathermy in these patients.

Maitland's case [82] illustrates the confusion that exists with regard to the classification and interpretation of certain tumors of the jaw, which often involve the maxillary sinus. The specimen in his case was diagnosed by three different pathologists as showing (1) adamantinoma, (2) chordoma and (3) tumor of the salivary gland. The growth in question involved the area about the upper molars, with invasion of the maxilla and maxillary sinus. Several operations were performed and followed up with roentgen therapy. When seen five years later, the patient displayed no evidence of any recurrence and roentgenograms showed reorganization of the bone with complete sclerosis of the maxillary sinus.

Wille [83] records a case of plasmacytoma of the upper jaw in a man 55 years of age who was treated with roentgen irradiation (10,400 r) and was clinically well after six years. He was the only patient with a lesion of this kind encountered at the Norwegian Radium Hospital in a series of 220 patients with malignant tumors of the nose and sinuses seen from 1932 to 1944. While plasmacytomas do not metastasize as frequently as other types, they are considered malignant because of a tendency to local recurrence and deep invasion. In growth and development they resemble other malignant tumors of the maxilla and the diagnosis can be made only on biopsy. Good photomicrographs and roentgenograms illuminate the article.

Heimendinger and Fruhling [84] report a case of reticular sarcoma of the maxilla in a man 33 years old that ran a fulminating course for six weeks, culminating in widespread metastases. The diagnosis was not made until late in the process, which had its onset after the extraction of an upper third molar. This was followed by swelling of the face and temporal region and symptoms suggesting infection. Continued swelling with orbital symptoms and hemorrhage from the nose and tooth socket led to the taking of biopsy specimens from several areas, which revealed the true nature of the process. Eventually exophthalmos, paralysis of the facial nerve and metastases in the lung, above the clavicle, in the prostate and in abdominal structures developed in rapid succession.

82. Maitland, G. R.: Atypical Adamantinoma of the Maxilla: Report of Case, J. Oral Surg. **5**:351 (Oct.) 1947.

83. Wille, C.: A Case of Plasmacytoma of the Upper Jaw, Acta oto-laryng. **36**:311, 1948.

84. Heimendinger, E., and Fruhling, L.: Considérations anatomo-cliniques à propos d'un cas de sarcome réticulaire des maxillares, Acta oto-laryng. **36**:323, 1948.

Biopsy specimens showed a highly cellular mass with little stroma, the cells assuming odd shapes, squat, angular and pointed, with nuclei irregularly staining in a network and protoplasm faintly basophilic. An occasional muscle giant cell was visible. The authors discuss nomenclature and decide that the classification of lymphoid neoplasms suggested by Géry and Bablet [85] would put their case in the category of reticular sarcoma.

Coen [86] reports a case of lymphosarcoma involving both maxillas in in child 5 years of age. Osteomyelitis complicated the situation and, despite three operations and irradiation therapy, the child died. The author presents a classification of lymphocytic tumors and discusses the histogenesis in this particular case. The diagnosis rested among lymphocytoma, lymphosarcoma and endothelioma. He decided that the growth originated in true lymphatic tissues. He feels that the osteomyelitic process about the roots of the teeth had something to do with stimulating the lymphocytic elements to develop into neoplastic formation.

An abstract of Torkildsen's report [87] follows:

A woman, 40 years old, had observed for about three years increasing ocular protrusion on the left side associated with periorbital edema. Ophthalmoscopy showed choked disk on the left side. A roentgenogram of the skull revealed sclerosis of the left sphenoid wing and of the left orbital roof. Meningioma en plaque was the diagnosis and the growth was partially removed. Recovery followed with improvement of the symptoms.

Meurman [88] cites Cushing concerning the incidence of meningiomas, comprising 13.4 per cent of all endocranial tumors. Other authors claim an incidence varying from 16 to 20 per cent. The tumors are most commonly noted along the lesser wing of the sphenoid and the olfactory groove. The author reports on 6 patients, 4 of them women. The average age was 42 years. Most of the tumors were quite large, almost filling the anterior fossa and originating from the region of the lamina cribrosa. Diminution of the olfactory sense was observed in 4 patients. Papilledema and atrophy of the optic nerve occurred in all of them. Mental disturbances were observed in 3 patients. Headache was also a common symptom. Roentgenograms in 5 revealed definite changes in the contour of the cribriform plate. Arteriography in 1 patient showed displacement of the carotid siphon. The anterior cerebral and ophthalmic

85. Géry, L., and Bablet, J.: Les néoplasmes malins des organes lymphoïdes: Essai de classification, Bull. Assoc. franc. p. l'étude du cancer **24**:673 (Dec.) 1935.

86. Coen, R.: Sui tumori linfosarcomatosi dei seni mascellari nell'infanzia, Ann. larying., otol. **46**:65, 1947.

87. Torkildsen, A.: Meningeoma of the Spenoid Ridge with Exophthalmos, Nord. med. **38**:1027 (May 21) 1948.

88. Meurman, O. H.: Meningiomas of the Olfactory Groove, Acta oto-laryng. 1948, supp. 67, p. 76.

arteries were also out of line. Relief in this condition can be obtained only via surgical intervention. The author describes the operations of Dandy and Tonni. Only 2 of the author's patients recovered.

Abdeen [89] presents photographs of a patient with a most extensive fungating carcinoma involving the orbit and ethmoid cells and forming a huge mass piled up externally over the eye, root of the nose, forehead and cheek. Since a radium bomb was not available, the author employed two tubes of 50 mg. of radium each hung in the dome of a wire cradle resembling an anesthesia mask 3 cm. from the surface of the lesion. The tumor was irradiated in segments for twenty-four hours over each segment, the procedure being repeated over and over again until 37 exposures had been made. At the same time five needles, each containing 2 mg. of radium, were placed in the left nostril in the ethmoid region for seven days. The lesion disappeared entirely and when the patient was last seen, one year later, there was no sign of recurrence. While admitting that a cure cannot be claimed because of the short time elapsed, the author comments on the effectiveness of the agent despite the small amount of radium available. The histologic diagnosis on biopsy was "alveolar carcinoma probably arising from one of the salivary or lacrimal glands."

A case of chordoma of the right frontal sinus (Adams [90]) is extremely rare, since chordomas are usually seen at the ends of the vertebral column. However, in this instance the appearance on sections seemed to conform to the accepted classification. The growth occurred in a 64 year old woman who had had right frontal headaches severer during the day and often of great intensity. After about a year she knocked her head against an oven door, after which an abscess developed medial to the right eye and was incised and drained. Roentgenograms showed sclerosis of the frontal sinuses and darkness in the maxillary sinus. The latter was opened and bone fragments and membrane removed for biopsy. At the same time the draining fistula in the frontal sinus was explored and tissue removed. The antral tissue was pronounced inflammatory, while the biopsy specimen from the frontal sinus showed a cellular mass, the cells being well defined with clear cytoplasm and hyperchromatic nuclei. The diagnoses of myeloma, chondroma and chondrosarcoma were considered but excluded, since the sections did not conform to their classic pictures. Despite intensive irradiation, the condition still persists, although there has been considerable improvement.

89. Abdeen, M. O.: Radium Treatment of Cancer of Orbit and Ethmoid Cells, Arch. Otolaryng. **47**:185 (Feb.) 1948.

90. Adams, W. S.: A Case of Chordoma of the Right Frontal Sinus, J. Laryng. & Otol. **62**:93 (Feb.) 1948.

Bjuggren [91] finds it difficult to decide whether his 70 year old patient (female) had osteitis deformans (Paget's disease) or von Recklinghausen's disease (generalized osteitis fibrosa cystica). The author presents a detailed history, including the results of thorough examination. The case was reported because of a solitary giant cell tumor removed from the inferior turbinate. There had been repeated nasal hemorrhages for ten years.

Myeloma (plasmacytoma) in Chase's case [92] gave rise to symptoms suggesting frontal sinusitis, osteomyelitis and possible carcinoma for several weeks before a biopsy specimen revealed the true nature of the condition. External operation disclosed necrosis of the superciliary ridge and the posterior wall of the frontal sinuses, which were filled with thick grayish brown syrupy material. The laboratory report was "plasma cell myeloma." Despite roentgen therapy and the administration of stilbamidine® (4,4'-diamidinostilbene), the patient died a few months later. Metastases were responsible for paralysis of the lower extremities and involvement of the humerus and pelvic bones.

Grégoire and Martin [93] claim to have found only 29 authentically reported cases of primary epithelioma of the frontal sinuses, to which they add 2 of their own. One was of a woman aged 28 who had been operated on for a cyst in the suborbital region in 1940, with recurrence in 1945. Recently she was treated for frontal sinusitis with repeated nasal hemorrhages. There were some external swelling and a small tender mass. Roentgenograms showed cloudiness and hazy outlines of the frontal sinus. The cystic mass was removed and was reported to be an *épithélioma malphigien baso-cellulaire*. It was active and infiltrating with pronounced stroma reaction and zones of necrosis. The patient was given a series of 14 roentgen treatments of 250 r each, with decided relief of her headaches and hemorrhages. The ultimate result was not mentioned.

The second case was that of a man of 42 who had headaches, sanguineous nasal discharge and a small tumor at the nasofrontal angle. Roentgenograms showed destruction of part of the anterior sinus wall. At operation the mass, which was gelatinous and hemorrhagic, was seen to have eroded the orbital wall, the posterior wall and part of the floor. It extended through the intersinus partition and into the ethmoid cells. The latter were thoroughly exenterated and a large passage was made

91. Bjuggren, G.: Solitary Giant Cell Tumor in Nose in Case of Ostitis Deformans Paget or Case of Ostitis Fibrosa Generalista Engel-Recklinghausen?, Acta oto-laryng. **36**:34, 1948.

92. Chase, W. D.: Solitary Myeloma Mistaken for Frontal Sinusitis, Arch. Otolaryng. **48**:244 (Aug.) 1948.

93. Grégoire, A., and Martin, P.: Deux cas d'épithélioma primitif du sinus frontal, Oto-rhino-laryng. internat. **32**:45 (March) 1948.

into the nose. The histologic report was *épithélioma malphigien intermédiaire à tendance baso-cellulaire*. This patient received the same amount of irradiation as the first, with a good primary result. The authors discuss the symptomatology under two heads, namely, the intrasinus phase and the phase of exteriorization, with points of differentiation from mucocele, osteoma, tertiary syphilis, tuberculosis and polyposis. They state that the prognosis is poor because of the rapid extension to the adjacent sinuses and because early diagnosis is frequently missed. The treatment, both surgical and by irradiation, is only palliative.

Friedberg [94] reports 3 cases of sellar tumors invading the nasal cavity, stressing the importance of the nasal examination in such cases and the significance of any polypoid growth encountered in the region of the olfactory fissure. One case was of a pituitary tumor in a woman who had dystrophy over a period of years. Although some regression was observed with irradiation therapy and the administration of hormones, the case ended fatally.

The second case was of adiposogenital dystrophy (Frölich's syndrome) in a young soldier. The patient underwent an osteoplastic craniotomy and a growth was removed from the sella and sphenoid sinus. Postoperative rhinorrhea was controlled by a subsequent procedure in which the defect was closed with a strip of muscle and fascia. The patient recovered, although reporting lack of energy and some "eye trouble."

In the third case, a pinkish gray mass was seen in the olfactory sulcus and in the nasopharynx. This patient, a woman, also underwent craniotomy and a mass, which seemed to be extradural, was removed in part from the apex of the orbital roof. Radon seeds were implanted. Subsequently, several attempts to reduce the intranasal mass by means of surgical diathermy reaching into the posterior ethmoid cells, sphenoid sinus and upper septum yielded only temporary improvement. However, the tumor seemed to grow more rapidly after each procedure, filling the nasal cavity and protruding from the nostril. Her condition deteriorated rapidly and a fatal outcome was anticipated. Several sections of the mass appeared chordomatous, while others were typically sarcomatous.

The case presented at a pathologic conference in the Massachusetts General Hospital, Boston, by Cogan [95] concerned a 65 year old woman who gave a history of sensitivity to heat, cold and pressure in the third right upper molar, followed by a watery discharge becoming bloody. The pain increased in severity and spread to the right temporal region.

94. Friedberg, S. A.: Intranasal Extension of Sellar and Parasellar Neoplasms, Laryngoscope **57**:347 (May) 1948.

95. Cogan, D. G.: Transitional Cell Carcinoma of Sphenoid Sinus, New England J. Med. **239**:837 (Nov. 25) 1948.

Roentgenograms at this time failed to disclose anything pathologic. However, in a short time, ptosis and diplopia appeared, ending finally in complete ophthalmoplegia. Roentgenograms now showed increased density in the right sphenoid sinus and the outline of the sella was not clear. There was also calcification of the carotid arteries. Results of spinal fluid studies were not helpful. Discussion follows concerning the reason for failure to make the diagnosis early, it being pointed out that careful examination with a nasopharyngoscope might have disclosed bloody discharge coming from the sphenoid sinus. The sphenoid sinus was explored and some tumor tissue removed; probing disclosed destruction of the roof of the sphenoid. The diagnosis was transitional cell carcinoma. At the time of the report the patient was undergoing roentgen therapy and was somewhat relieved of her severe pains.

Tod[96] presents a résumé of results obtained in 222 patients with cancer of the maxillary sinus treated at the Holt Radium Institute, Manchester, England, in a ten year period. The patients with "early" cancer numbered 95, of whom 29 were well at the end of five years (30 per cent). The growths in 127 patients were labeled "late"; 17 of these survived the five year period apparently cured. Their treatment represented several different technics, some of which also included surgical therapy. However, a group of 100 patients was treated by the simple procedure of inserting a 25 mg. tube of radium covered with thin rubber directly into the center of the growth and allowing it to remain until a total dose of 8,000 to 10,000 r was delivered at a point 2 cm. from the central radium source. The extent of the growth was determined by roentgenograms in various projections, and the tumor penetrated through a sublabial incision, through the palate or directly through the face. Since the tube was in situ seven to nine days, infection usually took place along with sloughing. This could be fairly well mitigated by the administration of penicillin. Subsequently it was necessary to enlarge the opening in order to remove necrotic tissue and dead bone. The results in this group of 100 patients were as follows: 38 per cent of the patients with "early" cancer and 13 per cent of the patients with "late" cancer survived the five year period. The author claims the results of this method are better than those formerly obtained by surgical procedures combined with irradiation and less distressing to the patient. However, mixed tumors treated by the combined procedure yielded 42 per cent of five year cures. Reticuloendothelial tumors, being more sensitive to roentgen rays, were treated by exposure to high voltage rays covering an area considerably beyond the limits of the growth.

96. Tod, M. C.: The Treatment of Cancer of the Maxillary Antrum by Radium, Brit. J. Radiol. **21**:270 (June) 1948.

BENIGN TUMORS

House [97] reviews a series of 30 cases of cysts of the superior maxilla, 24 of which involved the maxillary sinus. Fifteen were of dental origin, namely, 6 follicular and 9 radicular. Six were secreting or retention cysts; 5 were nonsecreting cysts of the sinus mucosa. Two were median anterior maxillary cysts and 1 was of the anterior wall of traumatic origin. The pathogenesis is discussed and typical case histories with accompanying roentgenograms and photomicrographs are presented. House points out that, while the follicular and radicular cysts both originate from dental embryonic rests, they differ strikingly in one respect, that is, the follicular cyst contains an unerupted permanent tooth whose crown is always directed toward the cyst cavity, whereas the radicular cyst develops around the root of an already erupted tooth. The symptomatology of cysts is not characteristic except in cases in wich they attain sufficient size to become apparent in the cheek, gingival recess or palate. The diagnosis is not difficult if the examination includes careful roentgenograpy, the use of contrast mediums and diagnostic puncture. The true origin and classification in some instances will not be made clear until after histologic examination of the excised specimen.

Kočka [98] describes a case of three mucous cysts of the left maxillary sinus which manifested themselves by severe headaches, localized at the back of the head and later at the forehead. On puncture of the sinus amber yellow liquid emerged. Transillumination and roentgen examination showed a shadow localized in the base and lateral part of the left maxillary sinus. The cysts were removed by a radical (Caldwell-Luc) operation. On histologic examination the mucosa of the maxillary sinus and the wall of the cysts showed signs of a healed localized inflammation of which only the scars remained. The wall of the cyst consisted of a row of cylindric cells. The fibrils were present but not in great numbers. Neither edema nor eosinophil leukocytes in the mucosa of the tunica propria were noted. It was a case of retention cyst of the mucous glands. Considering that the changes caused by the inflammation were not great and because no constitutional and allergic manifestations were observed, the author was of the opinion that besides the changes of inflammation there must be another cause. Constitutional disposition resulting from repeated infection of the upper respiratory tract can only be supposed.

Aschan [99] reported round soft tissue shadows in 92 maxillary sinuses of a total of 1,200 examined by roentgenography. Specimens from 17

97. House, L. R.: Benign Cysts of Superior Maxilla with Special Reference to Cysts of Maxillary Sinus, Arch. Otolaryng. **48**:301 (Sept.) 1948.

98. Kočka, Z.: Etiology of Mucous Cysts of Maxillary Sinus, Casop. lék. česk. **87**:438 (April 13) 1948.

99. Aschan G.: Ueber isolierte, zystisch veränderte Kieferhöhlenpolypen (Pseudozysten), Acta oto-laryng., 1948, supp. 74, p. 297.

were examined histologically after surgical treatments. Only one of these revealed a retention cyst with epithelial lining and covering. The rest were considered to show cystic degeneration of simple polypi. Eosinophilia and positive skin reactions revealed the allergic nature of these cases. For this reason the author does not recommend surgical therapy in most such cases. He would except, however, those cases in which the cyst was large enough to block the ostium and cases of aviators. He prefers the Caldwell-Luc operation to an intranasal antrostomy, having encountered several recurrences after the latter procedure.

Kristensen [100] reports a case of mucocele in a 30 year old woman which was first noted when she was 17 years of age. At that time operation confirmed the diagnosis of a meningoblastoma developing in the region of the anterior clinoid process. Several months later the patient was admitted to the ophthalmologic service with an exophthalmos caused by a tumor of the frontal sinus. A meningoblastoma associated with mucocele was the diagnosis and operation was performed. Twelve years later the patient was again seen with an extensive recurrence of the growth; both mucocele and meningoblastoma were observed, but the patient finally died of meningitis and cerebral edema. The case is reported because it was apparent that the mucocele was the result of obstruction of the duct by the ossifying growth.

Newlands [101] classifies cysts of the maxillary sinus as those arising from the sinus mucosa and those originating in the jaw or the teeth. The commoner types of the latter origin are the follicular and the dentigerous cysts, an example of each being presented in detail. Other types described are the developmental cysts, such as the median palatine, incisive canal and globulomaxillary cysts. Other inflammatory processes and bone diseases are mentioned because they frequently are mistaken for cysts in the diagnosis. Essentially a follicular or radicular cyst is the result of an inflammatory process at the root of a tooth, whereas the dentigerous cyst develops from the dental follicle or tooth sac.

The author discusses methods of approach in treatment of these conditions. He favors dealing with cysts involving the antrum by means of the Caldwell-Luc procedure, with sufficient exposure to facilitate complete removal of the mass and the creation of an adequate nasoantral window. If the cyst is in close contact with the floor of the sinus, it may

100. Kristensen, H. K.: Mucocèle du sinus frontal consécutive a un méningoblastome ossifiant, Oto-rhino-laryng. internat. **32**:113 (July) 1948.

101. Newlands, W. A.: Benign Cysts of the Antrum, Originating from Jaw or Teeth, Laryngoscope **58**:402 (May) 1948.

be advisable to leave a portion of its wall intact rather than risk the formation of a fistula into the mouth or interference with the blood supply to the teeth.

Fox and Berger [102] state that most cysts of the maxillary sinus come under the head of radicular or dental root cysts, follicular cysts or adamantinoma. The diagnosis rests on roentgenographic appearance and results of biopsy and aspiration. One case of radicular cyst filling the antrum is cited.

The case reported by Terracol and Fabre [103] is interesting because of the youth of the patient (14 years). An infected frontoethmoid mucocele caused the usual symptoms of exophthalmos and displacement of the bulb, in addition to the external signs of inflammation. Roentgenograms showed the extent of the mass, the absorption of some of the paper plate and the thinning of the floor and anterior wall of the frontal. The operative observations confirmed the diagnosis and a good result was obtained.

Pesti [104] contends that trauma plays but a minor role in the pathogenesis of mucoceles. He believes that anatomic factors form the true basis for the development of this condition. The fact that the overwhelming majority of these masses are to be found in the frontal sinus and anterior ethmoid cells seems to support his thesis, because here the communication with the nose is by way of a narrow tortuous duct often measuring only a fraction of a millimeter in diameter. It is conceivable that such a passageway may become occluded as a result of an inflammatory process for a sufficient length of time to favor the retention of secretions. The comparative rarity of mucocele in the maxillary and sphenoid sinuses, despite the frequency with which they are subject to infection, is explained on the basis of their more direct communication with the nasal meatuses through ostiums of definite caliber which, though temporarily blocked by inflammatory process, are rarely occluded for any extended period.

Larroudé [105] presents an interesting case of bony tumor involving the nose and sinuses in a 24 year old patient. It appeared at first in the left nasal fossa and was removed via lateral rhinotomy. Histologically it was diagnosed as an osteoma with cartilaginous inclusion. Eight months later the tumor reappeared in the ethmoid region, growing

102. Fox, S. L., and Berger, A. A.: Cysts of the Antrum, Eye, Ear, Nose & Throat Monthly **27**:468 (Oct.) 1948.

103. Terracol, J., and Fabre, L.: Mucocèle fronto-ethmoïdale de l'adolescence, Oto-rhino-laryng. internat. **35**:25 (Feb.) 1947.

104. Pesti, L.: Ueber Pathogenese der Mukocelen von anatomischem Gesichtspunkt, Acta oto-laryng. **36**:481, 1948.

105. Larroudé, C.: Contribution a l'étude histo-pathologique des tumeurs osseuses du nez et des sinus, Acta oto-laryng., 1949, supp. 78, p. 100.

slowly, and at the same time a new growth was seen in the canine fossa, which appeared to be developing more rapidly. Several months after the third operation the patient presented an enormous tumor invading all the cavities of the face, which was removed by bilateral resection of the two superior maxillas. Six weeks later a new tumor was noted adherent to the cribriform plate, which was operated on and from which the patient died. The author shows several photomicrographs demonstrating the different types of structure of the various tumors removed. It appears that while in the beginning the growth was purely a chondro-osteoma it later showed a more mixed configuration comprising fibrous, myxedematous tissue, along with bone and cartilage. Eventually the specimens showed sarcomatous degeneration. He calls attention to the calcareous bodies found in the specimens, which he thinks must play a role in the development of these malignant tumors about the sinuses.

Brunner and Spiesman[106] present 4 interesting cases of osteoma of the frontal and ethmoid sinuses, with complete and detailed analysis of the microscopic findings. One of the growths was associated with a large pneumocephalus. The clinical course of these cases is given in some detail and makes interesting reading. Among the conclusions reached are the following:

1. Small tumors may cause no trouble and remain quiescent for years, particularly in persons over the age of 50.

2. The increase in the size of an osteoma is due to metaplastic ossification of the connective tissue and the deposit of new bone by the paranasal mucosa, which serves as a periosteum.

3. The mixed type of stroma, that is, spongy and compact bone, is the most frequent form encountered.

4. There are some points of similarity between osteoma and osteitis deformans. The difference, however, lies in the type of bony trabeculae, which in osteitis deformans present a mosaic structure, whereas in osteoma they consist simply of lamellar or woven bone. In the former one finds many more osteoblasts and osteoclasts than in osteoma. Osteoma is more likely to be pedunculated, which is rarely the case in osteitis. In osteitis one will frequently discover that other areas of the skull are involved. Furthermore, Paget's disease occurs in older persons than does osteoma.

In Velasco's case[107] the osteoma was situated in the lower part of the frontal sinus, blocking the duct and leading to an infection which produced pain, redness and swelling externally. This complication was controlled with antibiotic therapy, after which the mass was removed

106. Brunner, H., and Spiesman, I. G.: Osteoma of the Frontal and Ethmoid Sinuses, Ann. Otol., Rhin. & Laryng. **57**:714 (Sept.) 1948.

107. Velasco, R.: Osteoma del frontal, Rev. otorrinolaring. **7**:121 (Sept.) 1947.

through an external incision. Adequate drainage into the nose was provided by exenteration of the ethmoid cells and a good result was obtained.

Lesions of the frontal bone simulating mucocele are not common. Simonton and Medwick [108] report 2 such cases. One growth was unquestionably a true primary cholesteatoma, since the cavity, which contained exfoliated epithelial masses and cholesterol, was lined with epithelium and was separated from the frontal sinus by a distinct layer of bone. In the other case the type of lesion was more obscure. Although the mass was definitely not connected with the sinus and contained blood pigment and cholesterol, it failed to show any evidence of an epithelial lining. The authors, however, feel that rather than the result of a minor trauma long since passed, the lesion was more likely a cholesteatoma in which a secondary inflammatory process had destroyed the lining.

Bjork [109] calls attention to the difficulty in differentiating between a nasopharyngeal fibroma and a true fibroma arising from the sphenoid sinus. A case observed by the author was that of a man 56 years of age, in whom symptoms of unilateral nasal obstruction led to an attempt at removal of a mass that was seen in the nose. The attempt failed because of severe hemorrhage. A month later another attempt at removal was more successful because of preliminary ligation of the external carotid. The mass was traced backward in the direction of the sphenoid sinus and was removed with a snare. The nasal passage was then seen to have been greatly distended by pressure of the mass, and the sphenoid ostium was easily visualized with the pedicle of the tumor projecting over its lower border. The mass measured 4.3 by 2.7 by 1 cm. Histologically it was shown to be true fibroma durum. The author reviewed the literature and found only one other similar case on record; this was reported by Manasse, and the diagnosis was later disputed by Eckert-Möbius.

The case cited by Proctor [110] was in a woman 56 years old who had had three operations on the nose, resulting in extensive adhesions between the septum and middle turbinate. A severe hematemesis brought the patient to the hospital, where the hemorrhage was traced to the ethmoid area previously operated on. The adhesions were separated and a friable bleeding mass was encountered. On biopsy this proved to be a "chromophobe adenoma of the pituitary gland" which had evidently

108. Simonton, K. M., and Medwick, J. X.: Epidermoid-like Lesions of the Frontal Bone Simulating Mucocele, Ann. Otol., Rhin. & Laryng. **57**:473 (June) 1948.

109. Björk, H.: Pedunculated True Fibroma of Nose Arising in Sphenoid Sinus, Acta oto-laryng., 1948, supp. 67, p. 9.

110. Proctor, D. F.: Invasion of Ethmoid Sinuses by Adenoma of the Pituitary Gland, Arch. Otolaryng. **48**:187 (Aug.) 1944.

eroded into the ethmoid capsule. Roentgenograms showed an enlarged sella with erosion of the base and fused clinoid processes. Roentgen therapy for three weeks, to a total of 3,600 r, resulted in resolution. At the time of writing (about one year later) the patient was asymptomatic.

SURGICAL TREATMENT

Gatewood and deVere [111] point out the advantages of the use of local anesthesia in surgical therapy of the sinuses: less bleeding, no danger of aspiration, cooperation of the patient, no nausea and vomiting and less surgical shock. They describe the location of the various sensory nerves supplying the nasal organ and sinuses, their points of exit from the skull, the areas they supply and the methods of blocking them. A number of illustrations helps materially to clarify the text.

Littel [112] points out that failure in surgical treatment of the sinuses may be the result of several factors, namely, improper selection of cases due to inadequate knowledge of the existing pathologic involvement, faulty selection of surgical technics, failure to recognize an allergic component and neglect in providing for proper nasal ventilation. He emphasizes the importance of complete submucous resection of the septum in order to provide the space for air currents to reach the olfactory meatus and aid in the ventilation and drainage of the posterior sinuses.

In a general review of the progress of otolaryngology, Flake [113] mentions early trephination of the frontal sinus in acute fulminating infections, followed by irrigation and possibly even opening of the anterior ethmoid cells, especially those about the opening of the frontal duct. He also mentions subdural empyema, citing Anderson's studies. The infection is transmitted by way of perforating veins, and its course may be short lived. Headache, stiffness of the neck, somnolence, Jacksonian seizures and hemiparesis are characteristic symptoms. Drainage is accomplished by irrigation through multiple enlarged burr holes in the frontal, temporal and parietal areas.

Bourguet [114] goes into considerable detail concerning the surgical anatomy of the ethmoid cells and frontal sinuses and presents many drawings showing various conformations and relationships, all by way of illustrating his ideas concerning the external operation. He discusses in detail the technic described by Mouret and that of Maduro both of

111. Gatewood, W. L., and deVere, C. A.: The New Technique for Local Anesthesia in Sinus Surgery, Laryngoscope **58**:848 (Aug.) 1948.

112. Littel, J. J.: Some Pitfalls in Sinus Surgery, Laryngoscope **58**:396 (May) 1948.

113. Flake, C. G.: Medical Progress in Otolaryngology, New England J. Med. **239**:819 (Nov. 25) 1948.

114. Bourguet, J.: Sinusites fronto-ethmoidales: Opération par voie antérieure et externe, Semaine d. hôp. Paris **24**:1161 (May 10) 1948.

which are similar to procedures in vogue in this country. He seems to favor removal of the anterior wall of the frontal sinus in preference to the floor and makes a strong plea for avoiding trauma to the duct. When the sinus is extensive, he advises an osteoplastic procedure.

Borasi [115] compares Pallestrini's technic with the procedures of Ogston and Luc, Taptas, Jansen and Jacques, Killian, Caliceti, Ledoux and Lothrop for the cure of chronic frontoethmoiditis. The chief difference lies in the preservation of the paper plate, ascending process and lacrimal bone—in short, all the external bony supports—destruction of which often leads to blockage of the drainage pathway. In the technic described, all the ethmoid cells are removed, together with the anterior end of the middle turbinate, so that the exenterated cavity communicates widely with the nasal cavity. Emphasis is, therefore, placed on removal of tissues internal to the diseased areas, rather than external as in the other procedures. The author presents some photographs of prepared specimens, together with a diagram showing the operation for unilateral and bilateral involvement.

Lieberman and Babb [116] removed the mucous membrane of the frontal sinuses in dogs, apparently by curettage, and subsequently (two weeks to four months later) reoperated and examined the resultant healed process. They discovered that the mucous membrane had failed to regenerate but had been replaced by a fibrotic tissue containing islands of bone and cartilage. Islands of mucous membrane occasionally seen were assumed to be due to incomplete exenteration. They conclude that these observations may explain poor results following radical surgical measures on these sinuses. Several excellent photomicrographs accompany the article.

Godsall [117] states that the only way to be sure that a chronic infection of the frontal sinus is permanently cured is completely to obliterate the sinus. This is done by curetting thoroughly and keeping the cavity packed until it fills with granulations which eventually consolidate. He performed this operation in more than 40 cases and reported the results to the British Medical Association in 1923. The author refers to Mosher's recommendation that the outer wall and floor of the sinus be removed and the soft tissues compressed by a pressure packing, which accomplishes obliteration but in a different manner. This seems to substantiate his position in the matter.

115. Borasi, G.: Il drenaggio nasale nel trattamento chirurgico delle sinusiti croniche fronto-etmoidali secondo il metodo Pallestrini, Oto-rino-laring. ital. **15**:248, 1947.

116. Lieberman, G. E., and Babb, J. W.: Nonregeneration of the Mucous Membrane of the Frontal Sinus After Its Surgical Removal (in the Dog), Arch. Otolaryng. **47**:421 (April) 1948.

117. Godsall, R.: Surgical Treatment of Frontal Sinus Infections, New Zealand M. J. **47**:268 (June) 1948.

Vernet [118] claims that most cases of acute frontal sinusitis can be relieved by free drainage of the antrum, and he recommends the use of the Watson-Williams rasp through the inferior meatus. He states that congestion in the middle meatus and the middle turbinate is thereby lessened, which facilitates drainage of the frontal sinus through the duct.

Harris [119] describes the use of a tantalum tube to maintain the patency of the nasofrontal passage after extensive surgical intervention in this region. The tube measures 5 by 7.5 by 10 mm. and is held in place for four to six weeks by means of no. 28 tantalum wire, which is drawn through the skin incision and anchored to the forehead by adhesive tape or a wire suture. Three cases are reported with appropriate illustrations.

Nosik [120] repaired a frontal defect in a case of extensive comminuted fracture of the supraorbital ridge with involvement of the frontal sinus. Since it was impossible to fit the many fragments of bone together after removal of the lacerated sinus mucosa, most of these were taken out and a tantalum plate 0.0007 inch in thickness "molded to conform to the orbital ridge over the frontal defect," with a posteriorly extended portion at right angles to form the orbital roof, was fitted tightly and retained by a single tantalum screw. The cosmetic result was satisfactory.

GENERAL NONSURGICAL THERAPY

Bumgardner [121] comments on the trend to conservatism in the therapy of sinusitis and cites his experience in cases of allergy due to the abuse of vasoconstrictors, citing 12 cases that were particularly obstinate. His procedure in acute cases is to shrink the nasal mucosa and, after determining the sinus involved, to cannulate the maxillary, frontal or sphenoid sinus, as the case may be, and to irrigate with a 1:5,000 aqueous solution of nitromersol (metaphen®), followed by the instillation of 1 to 2 cc. of penicillin (250 units per cubic centimeter). The treatment is repeated in two to four days, depending on the character of the discharge. Seventy-five per cent of the cases of acute maxillary sinusitis cleared after the fourth treatment.

Roberts [122] traces the progress in the therapy of acute sinus infection to the present and outlines the procedures he now employs. He has

118. Vernet, M.: Drainage du sinus maxillaire à la rape de Watson-Williams comme traitement systématique de la sinusite frontale aiguë, Bull. et mém. Soc. méd., Paris **152**:14 (June) 1947.

119. Harris, H. E.: The Use of Tantalum Tubes in Frontal Sinus Surgery, Cleveland Clin. Quart. **15**:129 (July) 1948.

120. Nosik, W. A.: Tantalum Cranioplasty in Fracture of the Supra-Orbital Ridge and Frontal Sinus, Cleveland Clin. Quart. **15**:125 (July) 1948.

121. Bumgardner, J. S.: Resume of Treatment of Acute Sinusitis, Kentucky M. J. **46**:271 (July) 1948.

122. Roberts, J. D.: Management of Acute Sinus Infection, Texas State J. Med. **44**:324 (Aug.) 1948.

observed the antibiotics to be of little avail when used locally and prefers to administer them intramuscularly. His experience with aerosol therapy has been too limited to admit of any conclusions. In the cases of maxillary sinusitis he prefers to avoid irrigation until the acute stage has subsided or at least until the temperature has receded to normal. The dangers attending early attempts at puncture are pointed out, and reference is made to unfortunate experiences in his own practice. Emphasis is placed on conservative treatment, such as use of shrinking agents, spot suction and intramuscular injections of penicillin. Acute ethmoiditis with abscess formation in children may necessitate external incision for drainage. External trephination of the frontal sinus is indicated in severe cases in which nasal drainage is inadequate.

Bergaglio [123] reviews recent trends in our conception of nasal physiology and the histopathogenesis of sinusitis. He believes that sinusitis is more frequent in children than it is generally believed to be and stresses the importance of recognizing its presence because of the possible relationship to chronic bronchial disorders, rheumatism and albuminuria. The therapy suggested follows the generally accepted modern procedures, with emphasis on the elimination of allergic factors, proper administration of antibiotics in acute infections and the rational use of vitamins.

Spearman [124] covers a wide variety of topics relating to the sinuses. These include anatomic, physiologic, pathologic and bacteriologic aspects, effect of physical and chemical agents, diagnosis, treatment and complications. His suggestions conform to recent modern trends and the article summarizes present day practices.

Van Alyea [125] exposes some of the commonly accepted concepts concerning the ethmoid region which he terms the "folklore of rhinology." The ethmoid bulla is generally not the cause of impaired frontal sinus drainage and seldom requires surgical correction. Ethmoid cells do not constitute one group but are definitely divided into anterior and posterior groups by the ground lamella of the turbinal. Infection in one group does not necessarily involve the other. Nasal polypi do not arise from the ethmoid cells but rather from the uncinate, the infundibulum, the margins of ostiums and the nasal surfaces of the ethmoid cells. Ethmoiditis rarely requires radical surgical treatment. Functioning structures should be preserved and drainage facilitated by freeing the middle meatus and eliminating contributing factors, such as allergy, gastrointestinal disturbances and endocrine and nutritional imbalance.

123. Bergaglio, O. E.: Rinosinusitis agudas y crónicas, Día méd. (supp.) **5**:45 (June 24) 1948.

124. Spearman, M. P.: Paranasal Sinusitis, Southwestern Med. **28**:9 (July) 1948; 17 (Aug.) 1948.

125. Van Alyea, O. E.: Nonsurgical Management of Ethmoiditis, Laryngoscope **58**:779 (Aug.) 1948.

DRUGS

Chen [126] discusses the various vasoconstrictors commonly used from the point of view of their chemical formation, as well as their pharmacologic action. Of the benzene-containing or aromatic amines, ephedrine still appears to be one of the best vasoconstrictors. Its methyl group on the α carbon atom from amine nitrogen serves as a protector against the action of oxidase. This is responsible for the prolongation of its action and its effectiveness when given by mouth. The only other product comparable to ephedrine is phenylpropanolamine (propadrine®) hydrochloride. Use of amphetamine is undesirable because of its stimulating action on the central nervous system. Among the straight chain or aliphatic amines, compounds of seven carbon atoms have optimal action. Thus, tuamine® and forthane® (methylhexamine) both have high potency. Here, the methyl group on the α carbon atom is again necessary to preserve the prolongation of action. A new compound, 2-methylamino-1-cyclopentyl-propane hydrochloride (no. 02040), which has a five-membered ring but also belongs to the aliphatic series, has been shown to be more active than tuamine,® methylhexamine and ephedrine. It is effective when given by mouth. It has one-fortieth the stimulating action of amphetamine on the central nervous system. Emphasis is placed on oral administration of vasoconstrictors instead of local application. This will eliminate those products which are not effective when given by mouth. The synergism of vasoconstrictors and antihistaminic drugs is also discussed.

Seydell and McKnight [127] report 8 cases in which anosmia or parosmia developed immediately or shortly after the use of a solution of tyrothricin in the nose by way of spray or drops. The disturbance was noted for four to eight months, and none of the patients fully recovered a normal sense of smell. Concomitant disturbance of the sense of taste was also noted. The solutions used were in concentrations of 1 : 5,000.

House and Carey [128] report a case of intoxication due to use of naphazoline (privine®) hydrochloride in a child 5 years of age. After the instillation of 2 drops of the solution in each nostril with temporary relief, the patient became drowsy and was difficult to arouse. This state persisted for several hours. Vague abdominal pains were complained of.

126. Chen, K. K.: Aliphatic Versus Aromatic Amines as Vasoconstrictors, Ann. Otol., Rhin. & Laryng. **57**:287 (June) 1948.

127. Seydell, E. M., and McKnight, W. P.: Disturbances of Olfaction Resulting from Intranasal Use of Tyrothricin: Clinical Report of 7 Cases, Arch. Otolaryng. **47**:465 (April) 1948.

128. House, L. R., and Carey, W. C.: Constitutional Effects from the Use of Sympathomimetic Drugs as Nasal Medication in Children: Report of Case of Privine Toxicity, Laryngoscope **58**:1294 (Dec.) 1948.

The next day 1 drop of solution was instilled in each nostril. Two hours later she was deeply asleep and hard to awaken, her pulse rate dropping to 50 and later to 44. The pupils were contracted; the carbon dioxide–combining power was 26 volumes per cent, and nonprotein nitrogen measured 31 mg. per hundred cubic centimeters. Otherwise no abnormalities were noted. The comatose state persisted for about thirty-six hours.

The authors refer to 4 similar cases previously reported in the literature and list the systemic effects of naphazoline hydrochloride as follows:

1. Prolonged hypertension with peripheral vasoconstriction.
2. Mild cortical stimulation, followed by depression.
3. Depression of basal centers — bradypnea, low temperature, tachycardia followed by bradycardia.
4. Resembles and potentiates the action of the barbitals in depression of respiration.

Furlong [129] treated 262 students for a total number of 670 colds, using par-pen® (a product providing, when freshly mixed, 800 units of penicillin per cubic centimeter in a 1 per cent solution of hydroxyamphetamine [paredrine®] hydrobromide) as a nasal spray twice daily. Patients with a total of 490 colds diagnosed as infectious, allergic or a combination of the two were closely followed. In 58 per cent the colds cleared up within one week and 88 per cent were discharged after two weeks' treatment. No unfavorable reactions were noted, and the author states that this preparation seemed the most satisfactory penicillin-vasoconstrictor combination with which he had had any experience.

Fineberg [130] successfully employed streptomycin locally in a case of chronic maxillary sinusitis of dental origin in a woman 30 years of age. The offending organism was identified as Alcaligenes faecalis. A solution of streptomycin, containing 2 mg. per cubic centimeter, was instilled into the sinus after preliminary saline irrigation by means of an indwelling catheter introduced through a large trocar via the inferior meatus. Instillations of the solution were done every twelve hours. At the end of three days the patient was free from symptoms. The following day a purulent discharge reappeared but yielded only Staph. pyogenes var. aureus. Irrigations with penicillin, 200,000 units in 5 cc. of saline solution, for a few days cleared up this secondary infection and the patient was free from symptoms when examined two months later. Roentgenograms also showed the sinus to be clear.

129. Furlong, T. F.: Clinical Test of a New Spray, Arch. Otolaryng. **48**:658 (Dec.) 1948.

130. Fineberg, N. L.: Streptomycin Used Locally in the Case of Chronic Suppurative Maxillary Sinusitis: Report of Case, Laryngoscope **58**:545 (June) 1948.

Fabricant [131] reviews recent trends in therapy of diseases of the nose and throat with regard to medicaments and states that inadequate dosage and indiscriminate use of antibiotics and sulfonamide compounds can breed resistant bacterial strains. "Their promiscuous use is unwarranted in trivial infections." The author calls attention to overmedication with nose drops and the fallacy of "alkalization" for acute respiratory infections. Also, the local use of antibiotics in acute conditions of the nose and throat is ineffective because the drug does not reach the deeper tissues where most of the bacteria reside. If chemotherapy is indicated, it should be administered parenterally in adequate doses. According to the author, vaccines have little value in the treatment and prevention of colds.

Davison [132] gave 50,000 units of penicillin every three hours intramuscularly to 50 patients with acute or chronic sinus infection with the following results: In the allergic group 11 with acute infections and 13 of the 18 with chronic infections were cured, the other 5 patients showing at least 50 per cent improvement. In the nonallergic group all 16 with acute infections and 4 of the 5 with chronic infections recovered. The allergic patients were so identified by the results of nasal smears and Hansel staining and were given diphenhydramine (benadryl®) hydrochloride or tripelennamine (pyribenzamine®) hydrochloride. The author emphasizes the importance of drainage, with irrigation of the antrums when indicated. Cultures from the sinuses showed Staph. pyogenes var. aureus in 16 patients, Streptococcus viridans in 11, hemolytic streptococci in 9, nonhemolytic streptococci in 8, Staphylococcus pyogenes var. albus in 7 and Pneumococcus in 4.

Miller [133] dramatizes the beneficial effects of penicillin mist in the treatment of sinus infections for the lay reader and credits the therapy far beyond its deserts. The title itself is provocative: "Relieves the Great American Headache—Sinus Trouble!" The author claims that 70 per cent of all subjects are benefited, basing her statement on the reports of Barach and associates at the Columbia-Presbyterian Medical Center, New York. While in the latter part of the article mention is made of possible drawbacks to the treatment, these statements are overshadowed by the exuberance with which the major part of the article is tinged.

Hollender [134] observes that penicillin is effective in treatment of the acute stage of sinusitis if given in adequate doses by the intramuscular

131. Fabricant, N. D.: Nasal and Throat Medication: A Survey of Current Trends, Laryngoscope **58**:793 (Aug.) 1948.

132. Davison, P. W.: The Penicillin Treatment of Sinus Infection, Pennsylvania M. J. **51**:272 (Dec.) 1947.

133. Miller, L. M.: Penicillin Mist Relieves Great American Headache—Sinus Trouble! Hygeia **26**:178 (March) 1948.

134. Hollender, A. R.: Nasal Sinusitis—Evaluation of Sulfonamides and Penicillin in Its Treatment, Eye, Ear, Nose & Throat Monthly **27**:414 (Sept.) 1948.

route, the degree of its efficacy depending on the sensitivity of the prevailing organisms. Its use should, however, be followed up by irrigation or Proetz displacement as soon as the acute symptoms have subsided. Medicated solutions have no advantage over isotonic sodium chloride solution for this purpose. In chronic infections antibiotic therapy may be helpful but cannot be depended on for cure. Aerosol administration of penicillin possesses certain advantages in selected cases but is not entirely without danger. In any event, it cannot be expected to exert much influence in chronic infections.

Monti [135] reports good results from the use of a sulfonamide preparation called pyridine N (*piridina N*) at the Instituto De Angeli. It is a nonirritating aqueous solution, stable, atoxic, readily absorbed, of p_H 7 and capable of dilution in any degree. The preparation is a 40 per cent solution of aminobenzene-sulfamidopyridine-methylsulfonate sodium and can be injected intravenously as well as intramuscularly. It is well tolerated locally in a 10 per cent dilution. He found it efficacious in the treatment of young children when administered locally by Proetz displacement. Some of these children not only were suffering from a profuse nasal discharge but presented otitic, pharyngeal and tracheobronchial involvement. Five adults with pansinusitis, 1 with dental involvement, were cured by this method. Among 6 persons with chronic purulent frontoethmoiditis, the therapy was successful in 3. The other 3 finally came to surgical treatment, although their condition definitely improved with the local treatment.

Faier [136] reports 11 cases of chronic sinusitis treated by a combination of local and systemic use of penicillin, with surgical intervention in several instances. The results were good when gram-positive organisms were found. In several of the cases the penicillin was employed locally at frequent intervals via an indwelling catheter through the inferior meatus. It is worth noting that in several of the cases improvement did not ensue until the penicillin was administered intramuscularly despite its previous employment locally. Irrigations were practiced at suitable intervals.

Gording [137] reports the results of treatment in 60 persons with acute and subacute sinusitis observed in 1946 and 1947. One-half the patients were treated with penicillin, the other half by the usual lavage with boric acid solution. Topical application of 3 cc. of a solution containing 2,000

135. Monti, P. C.: Sulla cura delle sinusitis nasali croniche con l'uso locale di antibiotici, Gazz. med. ital. **106**:267 (Dec.) 1947.

136. Faier, S. Z.: Treatment of Chronic Sinusitis with Penicillin, Arch. Otolaryng. **48**:507 (Nov.) 1948.

137. Gording, R.: Penicillin Therapy of Acute and Subacute Empyema of Antrum, Nord. med. **39**:1603, 1948.

units of penicillin per cubic centimeter two or three times daily, combined with intramuscular injections of 10,000 to 20,000 units, was used. The doses in several subjects were increased to 50,000 and 100,000 units, respectively. There was no difference in the results of treatment in the two groups.

Several patients whose sinusitis was associated with fever and other complications, and in whom ambulatory treatment had failed, were given continuous systematic treatment with penicillin. Satisfactory results were obtained in these patients. This may indicate that the doses originally used were too small. In addition to topical instillations, the author, therefore, has adopted the use of intramuscular injections of 300,000 units of penicillin in oil and wax. The results obtained with this treatment have been satisfactory, but the number of cases is still too small for publication.

Krasno, Karp and Rhoads [138] report gratifying results from the inhalation of penicillin dust in a series of 357 patients, of whom 174 were hospitalized. A simple apparatus is described by which 100,000 units can be inhaled in three to six minutes. Such treatments were given three times daily at first and later less frequently, depending on the severity or chronicity of the patient's condition. Cultures showed rapid disappearance of bacteria within a few hours after a treatment, and blood determinations revealed a penicillin concentration of 0.16 unit within an hour, dropping off slowly to 0.04 unit at the end of five hours. In 169 patients with infection of the upper respiratory tract, including common colds, 42 per cent were considered cured and 38 per cent 3 plus improved within three days. Pronounced subjective relief was noted. Allergic reactions were observed in 3 to 6 per cent of all subjects and were practically all of a local nature.

Payne [139] discusses the action of penicillin and the methods of administration and cites several cases in which the therapy was effective. These included a furuncle of the nose with symptoms of cavernous sinus thrombosis, acute exacerbation of chronic mastoiditis, acute laryngotracheitis, deep abscess of the neck and gas bacillus infection in a fractured left frontal bone. He concludes that the drug is successful mostly when given in sufficient doses to maintain high blood levels against sensitive organisms in the early acute stages of the infection. Nevertheless, one must be on guard against the masking of symptoms. It must always be borne in mind that penicillin is no substitute for surgical procedures when indicated.

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138. Krasno, L.; Karp, M., and Rhoads, P. S.: Inhalation of Penicillin Dust, J. A. M. A. **138**:344 (Oct. 2) 1948.

139. Payne, V. L.: Penicillin in the Treatment of Diseases of the Ear, Nose, and Throat, Laryngoscope **58**:1216 (Nov.) 1948.

Kenézy [140] submits a preliminary report on a new method of treating nasal polypi. These being generally allergic in origin, he proposes to desensitize the patient by means of an allergen obtained from the polypi in an ether-alcohol extract. This solution has been observed to elicit a positive skin reaction in 60 per cent of the cases. It is assumed that the allergen is a proteose and that the autolysate, when administered parenterally, will give rise to the production of tissue lysins which will cause the polypi to disappear. An extract obtained from polypi on one side of the nose has caused the polypi on the other side to disappear.

Hünermann [141] proposes use of a cod liver oil solution for instillation into the maxillary sinus in acute and subacute infections instead of the conventional irrigation. He claims a threefold benefit: First, if secretion is present it will be displaced by the solution and be available for diagnostic study; second, the size of the antrum is determined by the amount of solution it takes, and, third, the infection is combated and the tissues restored to normal. The treatment may be repeated if necessary after eight days, and if in some cases the process is not halted he advises intranasal antrostomy. The author claims that this therapy also exerts a beneficial effect on the tracheobronchial infection which frequently accompanies the sinusitis.

Gordon [142] attempted an evaluation of the respective merits of three antihistaminic drugs in the treatment of acute infections of the upper respiratory tract in several hundred persons. The subjects were divided into the following groups: those with bacterial infections, those with viral infections and those with infections complicated by an allergy. Administration of nose drops was avoided except when the patient used them on his own. The author's conclusions were that use of these drugs is helpful in making the patient more comfortable and in shortening the course of the episode. This was found to be the case in more than 85 per cent of the patients. Tripelennamine hydrochloride was the most reliable of the three drugs employed.

Taylor and Foter [143] made a number of tests with vonecidin,* a decongestant containing an active nontoxic antibiotic derivative and a quaternary ammonium germicide, showing its low surface tension and p_H value within normal physiologic range. It was found to be germicidal

140. Kenézy, T.: New Procedure in Therapy of Recurring Nasal Polyposis, Acta oto-laryng. **36**:421, 1948.

141. Hünermann, T.: Die Lebertranbehandlung der Kieferhöhlenentzündung, Acta oto-laryng. **36**:447, 1948.

142. Gordon, J. S.: Antihistaminic Drugs in the Treatment of Upper Respiratory Tract Infection, Laryngoscope **58**:1265 (Dec.) 1948.

143. Taylor, B., and Foter, M. J.: Bacteriological Studies on a Nasal Decongestant Containing the Antibiotic Methylol Gramicidin, Laryngoscope **58**:1206 (Nov.) 1948.

to a large variety of bacteria in the laboratory and to possess self-sterilizing properties.

Popa [144] recommends the insufflation of acetylsalicylic acid crystals or powder into the antrum for the cure of chronic sinusitis. This is done on four successive days following irrigation, and when successful it will evoke a profuse discharge of mucoid or mucopurulent secretion, which completely disappears after the conclusion of the series. If the treatment fails to provoke this secretion of mucus, it is considered a failure and the patient will have to be operated on. In fact, the author claims that this method of treatment not only will cure a certain number of cases of chronic purulent sinusitis but also serves as a criterion for surgical indication. He explains the action of the acid as inhibiting the growth of bacteria, by lowering the p_H of the secretions and by the temporary increase in the activity of the mucous glands.

Smith [145] goes into the causation of sinusitis, listing endogenous, exogenous and exciting causes, which include a wide variety of items too numerous to mention. Among 1,000 patients examined roentgenologically 40 per cent showed structural nasal deformities, such as narrowing of the nares, deviated septum, hypertrophy of the turbinals and high palatine arch. He makes much of the overpublicized milk diet, which is a poor substitute for food requiring the more active mastication so essential to the development of the jaws, the teeth and the palate. Often items mentioned as contributing to sinusitis are indulgence in the use of nose drops, nasal douching and swimming. The author also discusses symptoms, diagnosis and therapy. Under the last-named heading he describes the effects of short wave therapy and hails it as a most important adjuvant. However, he stresses the importance of eliminating causative factors and mentions the use of mild vasoconstrictors and vitamins for selected patients. Antibiotic therapy is of value in acute and subacute conditions but has little advantage in chronic infections. Several illustrative cases are cited.

GENERAL CLINICAL OBSERVATIONS

Putney [146] presents a brief review of the physiology of the sinuses and of the symptoms resulting from disturbances in normal function. He considers sinusitis under the headings of allergic, mechanical and bacterial, with some notes on orbital complications and osteomyelitis. The therapy suggested in these divisions is standard and conservative,

144. Popa, N.: Le traitement local des sinusites maxillaires par l'acide acétylsalicylique, Ann. d'oto-laryng. **65**:504 (Aug.-Sept.) 1948.

145. Smith, W.: Sinusitis and Short Wave Therapy, M. J. Australia **35**:640 (May 22) 1948.

146. Putney, F. J.: Nasal Accessory Sinus Disease, M. Clin. North America **31**:1344 (Nov.) 1947.

with emphasis on removal of anatomic malformations, improvement in drainage and judicious use of penicillin, particularly in the acute phases. Surgical treatment is indicated only when irreversible changes have been proved, when there is resistance to conservative treatment or in the presence of threatening complications. Instillation of penicillin (500 units per cubic centimeter) is recommended in treatment of several of the chronic bacterial types of sinus disease, with some measure of success despite the brief contact with the infected areas.

Gammage [147] touches briefly on the anatomy and physiology of the sinuses and attributes most sinus disease to the common cold. The causation is discussed, the influence of allergy given due credit and the therapy outlined along conservative lines.

MAXILLARY SINUS

Robin [148] reports a case of tertiary syphilis of the upper jaw, with necrosis of bone and two external draining fistulas. The man had been treated for syphilis twenty-five years previously and was free from symptoms in the interim. The early symptoms were painless swelling in the region of an upper molar, which was removed without relief, followed by perforation and discharge. Irrigation of the cloudy antrum showed normal results.

Work [149] reports an unusual complication of acute maxillary sinusitis following swimming in a boy 15 years of age. The symptoms came on about a week after the onset of the infection and consisted of elevated temperature, chilly sensations, paresthesia of the face, edema of the lids, trismus, engorgement of retinal vessels and presence of Babinski and Kernig signs. An abscess developed in the hard palate medial to the upper molar, which yielded pus on incision with some relief. Later another swelling was observed lateral to and above the molar in the region of the tuberosity of the maxilla. This also yielded pus and admitted a probe into the pterygomaxillary space. Instilled iodized oil found its way into the antrum. It could not be determined whether the infection reached the space by way of a dehiscence or through a necrosis of the posterior wall of the sinus. The patient recovered.

Tuberculosis of the sinuses is rare, only 27 cases having been previously reported from the literature by Myerson.[150] Hara and

147. Gammage, F. V.: Sinusitis, Virginia M. Monthly **75**:301 (June) 1948.

148. Robin, J. L.: Syphilis et osteite du maxillaire superieur, Rev. de laryng. **69**:187 (March-April) 1948.

149. Work, W. P.: Abscess of Pterygomaxillary Space Following Acute Suppurative Sinusitis: A Case Report, Laryngoscope **58**:1318 (Dec.) 1948.

150. Myerson, M. C.: Tuberculosis of the Ear, Nose, and Throat, Including the Larynx, the Trachea, and the Bronchi, Springfield, Ill., Charles C Thomas, Publisher, 1944.

Crane [151] discuss the pathogenesis and pathologic aspects in reporting a case of a woman 32 years old apparently cured with streptomycin treatment. There was a long train of symptoms for eighteen years, culminating in bilateral pulmonary tuberculosis which was treated in a sanatorium. Later profuse postnasal discharge and osteomyelitis of the mandible following a tooth extraction appeared. Antral irrigations were unsuccessful, and, when roentgenograms with contrast medium showed extensive pathologic involvement, a Caldwell-Luc operation was performed. The antrum was lined with thickened mucosa and contained much granulation tissue and pus. A fistulous tract was seen leading to the socket of the third molar. This was closed by a gingival flap and the sinus was drained through the nose by way of a catheter. The patient was given 2 Gm. of streptomycin intramuscularly in divided doses for ten days, and the antrum was irrigated twice daily with a solution containing 0.25 mg. of streptomycin. The fistula healed, discharge ceased and the patient was apparently relieved four months later, when she died as the result of a heart attack. Histologic examination of the excised tissue showed round cell infiltration, typical epithelioid cells and giant cells.

Rubin and Kling [152] cite several cases of sarcoid reported in the literature and present a case of their own, that of a 52 year old Negro woman. There was a history of progressively increasing nasal obstruction over a period of years, with the development of a tumor-like swelling in the septum at the level of the middle turbinate extending high up toward the vault. Submucous resection and middle turbinectomy were performed, but the mass recurred and eventually infiltrated through the nasal vault, causing discoloration of the skin and edema of the lids. The septum was swollen and appeared fused to the turbinal. There were much crusting and a tendency to bleeding. Roentgenograms showed areas of erosion in the nasal bones. The pathologist's report stated that the biopsy specimen showed "clusters of tubercle-like nodules without central caseation. The tubercles contain epithelioid cells, fibroplastic elements and round cells. Diagnosis: Chronic granuloma, consistent with sarcoidosis." At the same time (almost two years after the first operation) a mass appeared in the proximal phalanx of the right fifth finger, which on roentgenogram showed porosis of the bone, widening of the shaft and cystlike areas of rarefaction. Over a period of twenty-four days 44 Gm. of streptomycin was given without any effect.

151. Hara, H. J., and Crane, W. E.: Tuberculosis of the Maxillary Sinus: Report of Case Treated with Streptomycin, Ann. Otol., Rhin. & Laryng. **57**:1077 (Dec.) 1948.

152. Rubin, H. J., and Kling, D. H.: Boeck's Sarcoid of the Nasal Vault, Ann. Otol., Rhin. & Laryng. **57**:1083 (Dec.) 1948.

Goldman [153] states that when a fistula into an antrum appears after extraction of a tooth the sinus is likely to become infected. Consequently, he advises prompt administration of penicillin and cautions against tampering with the fistula. If small, it will close spontaneously. If large, it should be closed by a plastic procedure without delay. When infection is present, it should be treated by irrigations through the nose. Many of these infections will heal spontaneously. If the fistula persists along with the antral infection, the indications are to clean out the antrum via creation of an inframeatal antrostomy opening, or a Caldwell-Luc procedure if the mucosa is badly diseased. Plastic closure of the fistula should not be attempted until a later date, after the sinusitis is completely healed. Concerning the management of dental cysts, unless they are small the author recommends complete exenteration through a Caldwell-Luc operation, with primary closure of the buccal wound and intranasal drainage.

Koch-Langentreu [154] goes into considerable detail describing the anatomic, physiologic and pathologic aspects, diagnosis and therapy of maxillary sinus disease for the benefit of members of the dental profession. Most of the material is standard textbook stuff. However, he does cover several items which are of interest to rhinologists as well as oral surgeons. Accidental perforation of the antrum in the extraction of a tooth is treated conservatively if the antrum is healthy: no probing; no irrigation. A small perforation will heal spontaneously if it can be protected. In some cases it may be advisable to close the perforation by suture. On the other hand, if the antrum is infected surgical drainage by the intranasal or transcanine route is indicated. If a root of a tooth is broken off, roentgenograms will be needed to determine its fate, since it may be lost in a large granuloma or cyst, it may become wedged between the bony floor and the mucosal lining or it may lie free in the cavity. Removal of the fragment should not be attempted via the alveolus; the antrum must be opened widely enough, either intranasally or via the Caldwell-Luc method. The author also discusses cysts of the antrum, with comments on the indications for the Partsch and the Caldwell-Luc procedures, the latter being preferred when the cyst is large or when the antrum itself is infected. Concerning the closure of alveolar fistulas, the author emphasizes the necessity of providing sufficiently wide mucosal flaps so that tension is avoided. He prefers a flap taken from the palate,

153. Goldman, J. L.: The Relationship of the Maxillary Antrum to Oral Surgical Problems of Dental Origin, Oral Surg., Oral Med. & Oral Path. **1**:859 (Sept.) 1948.

154. Koch-Langentreu, J.: Die Kieferhöhle als Behandlungsgebiet des Stomatologen, Ztschr. f. Stomatol. **45**:201 (May) 1948.

its base being wide enough to insure good blood supply as well as mobility. The author states that it is not always possible to define sharply the limitations of the dentist and the rhinologist in connection with antral disease. Both must be fully cognizant of the close relationship between the teeth and the antrum. In any event, the surgical procedures are in the main rhinologic, although from the etiologic point of view a knowledge of dental pathology is essential.

Bourgoyne [155] likewise discusses certain antral conditions which are of interest to the oral surgeon. Oroantral fistulas resulting from improper extractions and postoperative care may be repaired in two ways: If not too large, the opening may be closed by relaxing incisions through the periosteum to the bone on each side of the socket. The periosteum is elevated, permitting approximation over the opening. The raw areas resulting from shifting of the mucoperiosteal bands will fill in with granulations that later cicatrize. If the opening is too large for this procedure, a palatal flap can be raised and swung over the defect. The author advises against attempts at removing roots or tooth fragments accidentally projected into the antrum via the tooth socket but suggests use of the direct approach through the canine fossa. In general, he advises dentists to familiarize themselves with antral lesions, such as infections, cysts and carcinoma, so that they may be in a better position to advise their patients, since dentists frequently are the first to be consulted because of dental symptoms.

Salazar Villanueva [156] cites 4 cases of odontogenic maxillary sinusitis in each of which the pathogenesis was different. In the first case an apical granuloma of a root which was in close proximity to the floor of the sinus caused an inflammatory reaction resulting in pain radiating through the branches of the infraorbital nerve. The sinus was cloudy and yielded some thin pus on puncture. Extraction of the root, penicillin irrigation of the sinus and local short wave therapy resulted in disappearance of all symptoms. Case 2 was one in which extraction of a premolar was followed by pain and tenderness. A fistula through the root socket into the sinus was demonstrated. Heavy doses of penicillin and short wave therapy brought about prompt closure of the defect without any serious involvement of the sinus. In case 3 the patient suffered excruciating pain in a second upper molar and, being far removed from a community where a dentist was available, attempted to extract the tooth with a forceps himself and succeeded in breaking it off, leaving

155. Bourgoyne, J. R.: Oral Contributions to Diseased Antrum, Oral Surg., Oral Med. & Oral Path. **1**:706 (Aug.) 1948.

156. Salazar Villanueva, R.: Sinusitis odontogénicas, Rev. méd. peruana **21**:403 (July) 1948.

part of the root in situ. This resulted in severe reaction and suppurative sinusitis with alveolar fistula. A Caldwell-Luc operation was necessary to clear up the infection. In case 4 an alveolar fistula followed the extraction of an upper molar and was treated by irrigation via the fistula for five months without effect. The condition cleared up after five puncture irrigations and intramuscular injections of approximately 5,000,000 units of penicillin.

Wakefield's article [157] is probably of more interest to the dentist than the rhinologist; yet there is much that the rhinologist can learn from it. He discusses the therapy in case of accidental puncture of the sinus floor through the tooth socket in the removal of a tooth, the loss of a tooth root in the antrum, fracture of the tuberosity during extraction and acute infection from abscessed teeth. He advises the least possible manipulation in the dental wound, preferring, if necessary, to open the antrum through the canine fossa for the recovery of a root and for the direct treatment of the infection. Misguided therapy through a tooth socket often leads to unnecessary widening of the wound, which predisposes to a permanent fistula necessitating a subsequent plastic closure, while often failing in its primary objective. Cleanliness, a minimum of local trauma and the use of a light dusting with sulfathiazole powder seem to be the essential guides in local therapy. The acute "red hot" infections must be treated conservatively despite the apparent urgency, lest premature surgical intervention lead to sinus complications.

FRONTAL SINUS

Crowe [158] states that a virus is the underlying cause of most of the conditions treated in the upper air passages. As a result, the way is paved for the entrance of pathogenic bacteria. The sinus cavities are shut off by swelling of the lining membranes, which favors retention and propagation of the bacteria. The nasopharynx is frequently the initial port of infection, particularly by way of the lymphatic elements. It is important, therefore, to see that spread in this tissue is controlled, and up to the present the best method of achieving this is with local irradiation. In acute frontal sinusitis the author has had good results from external trepanation through the floor, followed by instillation of penicillin or tyrothricin. In a series of 102 patients, 88 had to have this procedure, the remaining 14 recovering with antibiotic therapy. The procedure not only affords the patient immediate relief but also permits rapid regression of the inflammatory process so that the tissues may

157. Wakefield, B. G.: Maxillary Antrum Complications in Exodontia, J. Oral Surg. **6**:51 (Jan.) 1948.

158. Crowe, S. J.: Etiology, Treatment and Prevention of Chronic Sinus Infection, Ann. Otol., Rhin. & Laryng. **57**:364 (June) 1948.

return to normal without being traumatized. This definitely prevents the development of a chronic condition.

Radzimiński [159] reports primary cholesteatoma of the frontal bone in a girl aged 18, which was activated to rapid growth by a blow over the forehead about four years prior to his examination. The growth had been developing for three years, accompanied with headache and, recently, swelling of the lids and displacement of the bulb. Roentgenograms showed an expanded area to one side of the midline which resembled a mucocele. At operation an eroded area of bone was observed, leading into a cavity filled with foul cheesy exudate consisting mainly of pearly masses. The lining was a smooth glistening membrane. The sinus was opened and discovered to be normal. Histologically, the exudate revealed masses of squamous cells concentrically arranged. In the absence of any evidence of infection of the sinus, the author concludes that the growth was truly a congenital cholesteatoma.

Bateman [160] describes the symptoms of acute frontal sinusitis under three headings, catarrhal, suppurative and fulminating, which differ only in degree and intensity of the symptoms. In the absence of fever, patients whose symptoms are short of those of suppuration may be ambulatory. In such patients treatment is a matter of establishment of adequate ventilation by use of decongestants and the relief of pain. Antibiotic treatment is not indicated unless fever or signs of suppuration are present. Surgical therapy is indicated only when threatening symptoms arise or conservative therapy fails to give relief. The author makes a point in advising a thorough appraisal of the entire nasal apparatus as soon as the patient has recovered. This is done to ascertain the anatomic and physiologic status for possible etiologic factors in recurrence of the condition or in predisposition to chronicity.

SPHENOID SINUS

Winter's case [161] was of a boy 7 years of age whose diabetes was suspected to be due to disease of the sphenoid sinus because of symptoms of chronic nonsuppurative sinusitis. Roentgenograms, however, showed no abnormality. Examination revealed postnasal discharge and large tonsils and adenoids. The child's condition improved materially after tonsillectomy, to the extent that the posterior pituitary (pituitrin®) injections he had been receiving were discontinued. The author reports the

159. Radzimiński, A.: Cholesteatoma Involving Frontal Sinus, Polski tygodnik lek. **2**:1147, 1947.

160. Bateman, G. H.: Acute Frontal Sinusitis, M. Press **109**:502 (Dec. 3) 1947.

161. Winter, J. O.: Diabetes Insipidus—Amelioration Following Tonsillectomy, J. Pediat. **32**:78 (Jan.) 1948.

patient's condition during the past year as being good, although the postnasal discharge persisted, but to a less degree than formerly. He suggests the addition of chronic infection of the upper respiratory tract with sphenoid sinusitis to the list of causes of diabetes insipidus.

Proetz[162] brings out several important points in the therapy of sphenoid sinusitis. These are based on an appreciation of the remarkable tenacity of the cilia in retaining their vitality and ability to function despite adverse conditions. It is, therefore, necessary to avoid the use of drugs which are known to inhibit ciliary activity, such as cocaine and epinephrine (adrenalin®). Since the cilia wave in the direction of the ostium, it is also important to avoid trauma in this region. Drainage is best effected by displacement with a 0.25 per cent solution of ephedrine or 2-aminoheptane sulfate. If surgical treatment is indicated, it should be limited to creation of an opening which need not be large but should be placed as close to the ostium as possible and only through the thinner bone, since openings in the thicker portions of the anterior wall granulate rapidly and close in a relatively short time. This procedure will take care of the vast majority of chronic infections. Naturally, when intracranial complications are present, or when one is dealing with cancer or other tumors, more extensive procedures will be necessary, depending on the indications.

Ball and Thackery's case[163] was of a woman aged 39 who was followed for two years until her death. She had severe diabetes insipidus which was partially controlled by injections of beta-hypophamine (pitressin®) tannate in peanut oil. All her sinuses were involved and the antrums were opened and drained through intranasal antrostomy openings. Autopsy revealed one sphenoid sinus lined with a thick membrane containing many plasma cells and lymphocytes. The pituitary gland was surrounded by a thick inflammatory mass of tissue which seemed to be compressing the gland. The posterior lobe was small and necrotic and the anterior lobe showed some fibrosis. The author believes the infection spread from the gland and cites several references to support his contention.

ALLERGY

Hansel, Black and Ashley[164] contribute a detailed survey of the role of allergy in nasal conditions, covering diagnosis, incidence, com-

162. Proetz, A. W.: The Sphenoid Sinus, Brit. M. J. **2**:243 (July 31) 1948.

163. Ball, K. B., and Thackery, A. C.: Diabetes Insipidus Due to Sphenoidal Sinusitis, Lancet **1**:637 (April 25) 1948.

164. Hansel, F. K.: Allergy in Otolaryngology, Laryngoscope **58**:652 (July) 1948. Black, W. B.: Allergic Investigation; History Taking, Skin Testing and Diagnosis of Otolaryngologic Patient, ibid. **58**:673 (July) 1948. Ashley, R. E.: Allergy in Otolaryngology: Allergic Management—Treatment, ibid. **58**:686 (July) 1948.

plications and therapy. Much of this material has been written on before, but never so completely as here. The relationship of allergy to sinus disease is described by Hansel, who discusses the indications and contraindications for surgical intervention. In many cases this is a complex problem, since so much depends on the thoroughness of the study and the adequate appreciation of the degree of infection so far as tissue changes are concerned. It is to be noted that the continued use of vasoconstrictors is decried and the advice is given that ephedrine-like drugs and antihistaminic compounds be administered orally. As for antibiotics, it is thought that their local application is of doubtful value but that in some instances they may be employed parenterally to advantage. Surgical therapy in the sinuses is recommended only when the pathologic changes can be determined to be of bacterial origin and irreversible. This calls for a high degree of diagnostic skill. Certainly, in any case every effort must be made to diagnose and eliminate the allergic factor. The details of how this should be carried out are well given by Black and Ashley in their portions of the symposium.

Guimãraes [165] reports the results of his experience with 35 cases of allergic rhinitis. Before presenting the cases, he gives a classification of allergic reactions of the nose and paranasal sinuses in which he points out the complications possible in simple rhinitis. The author analyzes the question of incidence of allergic rhinitis, presenting data in relation to allergic diseases in general and to respiratory allergy. He also includes tables dealing with the symptomatology observed in his cases and the incidence of various allergens, emphasizing the importance of bacterial allergy and analyzing the results of treatment in relation to the duration of disease prior to beginning of therapy. Finally, the author draws attention to the occurrence of allergic rhinitis in persons having septal deflection or other anatomic deformity. Such a situation must be made clear before any therapeutic surgical procedure is indicated.

Larroudé [166] attempts to differentiate allergic sinusitis from chronic catarrhal or purulent types by means of roentgenologic studies with contrast mediums. The matter is quite simple when there is a definite history of allergy and when the elimination of the contrast mediums takes place within the normal time limit. However, difficulties arise when the case is of infection combined with allergy. Here the elimination time is prolonged, and, if the case is one in which there is little or no secretion, the examiner may have trouble determining which of the

165. Guimãraes, N.: Alergia naso-sinusal: Contribuçăo ao estudo clínico da rinite alérgica, Hospital, Rio de Janeiro **33**:749 (May) 1948.

166. Larroudé, C.: Sinusites chroniques et allergie, Acta oto-laryng. **36**:251, 1948.

two etiologic factors is dominant. The author states that many of these cases, often termed "silent sinusitis" because of the absence of local symptoms, are the cause of constitutional disorders and infections elsewhere in the body. In a number of the cases cited an operation was performed and extensive pathologic involvement uncovered in the mucosa. The author agrees with Hansel that an allergic stage may predispose to the introduction of infection, with consequent change in the whole picture. In this respect he differs with Robison, who claims that secondary hyperplasia in these allergic cases is the result of an evolution in which fibroblasts take over, causing reduction in the edema.

Kern [167] found 100 patients with perennial allergic rhinitis among a series of 516 persons with allergy of the respiratory tract of all types. Asthma was present in 246 and seasonal allergies in 170. The author states that the perennial nasal allergies were the most neglected of all. More than half the patients had suffered the complaint for more than five years before diagnostic skin tests were done. A number of these might never have been investigated were it not for complicating allergies, such as asthma, migraine and urticaria. In at least a third of the author's patients the nasal allergy was the forerunner of asthma often mistakenly diagnosed as "intrinsic." Inhaled allergens were observed to be the commonest offenders, with house dust, bedding materials, wool, domestic animals and pyrethrum heading the list.

The author discusses the value of skin tests and calls attention to the importance of proper interpretation. Patients often acquire new sensitivities, losing the old ones, so that repeated tests may be required. Treatment is based on recognition of the offending allergens and embraces avoidance of these items and desensitization. Nasal obstructions should be corrected and mild shrinking agents used to facilitate drainage. The author suggests radical surgical therapy when the sinusitis fails to respond to therapy after two months. The Caldwell-Luc operation is most effective but should be followed by careful after-treatment to prevent synechias and reinfection.

González Diaz, Angoloti de Cárdenas, Peñarrocha Alvarez and Jiménez Rodríguez [168] offer an extensive discussion on allergy, quoting liberally from the literature. They write on allergy in general, the allergic state, allergy of the respiratory system, the role of the sympathetic and parasympathetic nerves in vasomotor control, sensitivity to cold and the various manifestations of allergy in the nasal apparatus.

167. Kern, R. A.: Perennial Allergic Rhinitis: The Most Important Respiratory Allergy, M. Clin. North America **31**:1375 (Nov.) 1947.

168 González Diaz, A.; Angoloti de Cárdenas, I.; Peñarrocha Alvarez, J., and Jiménez Rodríguez, A.: Concepto local y focal de la alergia nasal y sinuso-paranasal, Medicina, Madrid (pt. 1) **16**:201 (March) 1948.

Diagnostic methods are gone into carefully, with emphasis on the eosinophil count and ways of determining it. Also mentioned are the various means of determining the offending allergen. The treatment outlined follows the usual trend toward local palliative therapy, including cauterization, and general measures divided into elimination or exclusion of the allergen and desensitization. The authors have noted Staph. pyogenes var. albus to be present in many cases and cite instances in which administration of autogenous vaccine helped materially in clearing up the symptoms. Twenty-four typical cases are cited.

TRAUMA AND FOREIGN BODIES

Seidel [169] advises conservative treatment during the first few days following gunshot wounds and fractures of the face involving the maxillary sinuses. As soon as the initial swelling has subsided, one must thoroughly examine the nasal chambers, reposition fragmented tissues, such as the septum and turbinates, and take measures to prevent subsequent synechias. Hematoma or abscess should be adequately emptied. The use of antibiotics has materially reduced the percentage of infections. Comminuted fractures should be reduced as soon as practicable, and loose fragments of bone replaced if they have even the slightest attachment to the soft tissues, since they so often retain their vitality and heal in. Massive trauma to the upper jaw causing numerous fractures and injury to the soft tissues requires an exposure as for a Caldwell-Luc procedure, so that the antrum may be examined, blood removed, bones replaced and provision made for drainage. Tooth roots projecting into such a cavity should be left alone, unless there is an infection in the adjoining bone. In many cases the teeth can be saved, but subsequent roentgenograms will be required to determine their status.

Lesions involving the sinuses at the base, namely, frontal, ethmoid and sphenoid, require particular care because of the danger of infection spreading to the meninges. A close watch on the temperature, pulse rate, blood picture and subjective symptoms is necessary to detect the onset of possible complications. Irrigations and other local therapy are contraindicated. Débridement of superficial areas is permissible, but extensive manipulations are contraindicated, unless there are signs of intracranial extension. Tetanus antitoxin and antibiotics should be given in every case.

Poinot and Dax [170] report briefly on 3 patients with fractures involving the frontal sinus, successfully treated by early exposure, removal

169. Seidel, O.: Ueber Verletzungen der Nase und der Nasannebenhöhlen sowie deren Behandlung, Chirurg. **17-18**:342 (May) 1947.

170. Poinot, J., and Dax: Trois fractures du crâne, intéressant les sinus frontaux, Mém. Acad. de chir. **74**:184 (Feb. 25-March 3) 1948.

of bone splinters, destruction of the sinus mucosa with the electrocautery, suture of the dura in one patient and closure by a fascial transplant in the other. In the third patient the posterior wall of the sinus was not injured. All the patients received penicillin in adequate doses.

The case of a foreign body in the posterior ethmoid cells reported by Chatellier and André [171] concerned a man 38 years of age who was violently thrown from his bicycle into a trimmed hedge. When first examined, he presented a prominent swelling of the lids and a horizontal linear wound less than 1 cm. in length just below the caruncle. The bulb was intact. Exploration of the wound uncovered a number of fragments of stems from the hedge. The wound suppurated and a fistula persisted which when probed led deeply to the posterior part of the os planum. An incision was made over the orbital plate beyond the caruncle and the fistulous tract followed to the posterior ethmoid cells. Only a restricted area of bony loss was encountered. A drain was inserted and penicillin administered. The wound healed and the patient went home. About two months later there was recurrence of fever and swelling and roentgenograms showed cloudiness in the ethmoid cells. The wound was reopened; the ethmoid cells were curetted, and a grayish mass was removed from the posterior cells in contact with the sphenoid.

Iubu, Surtea and Lohan [172] report a case in which a needle used to puncture the antrum of a 40 year old woman broke off during the procedure. All attempts to remove it through the inferior meatus were unsuccessful, and the authors were finally forced to do a Denker operation. The needle was observed to be wedged into the sinus walls and had to be broken in half before it could be extracted. The authors discuss the various difficulties that may arise in an attempted antral puncture, such as unusually thick bone, a small antrum, an antrum with septums, a thick membranous lining and cystic contents, all of which may predispose to complications. They express the necessity of considering the construction of new types of instruments for this procedure.

In the case reported by Salinger [173] a middle-aged woman was stabbed in the eye with a pocket knife and was brought to the hospital with the weapon still in situ. Roentgenograms showed the knife blade traversing the orbit medial to the bulb and downward into the maxillary sinus, the point coming to rest in the floor of the sinus near the posterior

171. Chatellier, H. P., and André, P.: Volumineux corps étranger de l'ethmoïde postérieur pénétré par effraction à travers l'orbite, Ann. d'oto-laryng. **64**:192 (March-April) 1947.

172. Iubu, M.; Surtea, S., and Lohan, T.: Aiguille de ponction brisée dans le sinus maxillaire extraitée par l'opération de type Denker, Rev. sc. oto-rhino-laryng. **11**:58 (Jan.-April) 1947.

173. Salinger, S.: Stab Wound of Orbit and Antrum, Arch. Otolaryng. **47**:503 (April) 1948.

border of the hard palate. Fortunately there was no danger to the bulb or the ocular muscles, and recovery was complete with minimal scarring to the upper lid.

MISCELLANEOUS

Meurman[174] examined 396 workers in lime and cement works to determine the effect of these substances on the sense of smell. The tests were conducted along the lines suggested by Proetz, differing only in the degree of concentration of the various solutions. Ten different substances were dissolved in paraffin in various degrees of concentration. The results were tabulated in olfacts, 1 olfact being defined as the smallest quantity of smelling substance (calculated in grams per liter of paraffin) which the normal nose is able to perceive. The results showed that the essential sense of smell lessened in direct proportion to the length of service at the works, being diminished in 21 per cent of those with under one year of service to 36 per cent of those with between five and ten years. Over half of all the subjects were noted to have a normal sense of smell. Lime dust was observed to be somewhat more harmful than limestone and cement dust.

The same author,[175] in a later publication, more extensive and in greater detail, presents the particulars of his investigations of 529 workers in lime and cement works for the purpose of determining whether the inhalation of these substances had any permanent deleterious effects on the respiratory mucosa, aside from the effect on the sense of smell alluded to in the previous paper. It was apparent that in the vast majority of workers, little or no effect could be noted so far as impaired function or increased morbidity were concerned. The incidence of acute infections varied with the seasons and ran parallel to that of the general community. Chronic changes were noted only in those persons whose exposure to the dust was prolonged over a period of years. It was, therefore, a question of degree of exposure whether a person could expect to suffer by reason of the exposure. This was further demonstrated by histologic studies of the mucosa of a number of white rats which were exposed to dust-laden atmosphere for varying periods. Changes in the mucosa, the olfactory bulbs and fibers were observed in these animals which were exposed for the longer periods. These consisted of increased secretion, thickened mucosa with dilatation of the goblet cells, swelling of the olfactory epithelium and, later, atrophy of the nerve fibers.

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174. Meurman, O. H.: Do Lime and Cement Dust Impair Sense of Smell? Acta oto-laryng., 1948, supp. 74, p. 313.

175. Meurman, O. H.: Studies of Effect of Lime and Cement Dust on Upper Respiratory Tract and Sense of Smell, Acta oto-laryng., 1948, supp. 73, p. 10.

D'Ajutolo [176] discusses pain in connection with sinusitis and shows how the location of the pain is not always in direct relation to the sinus involved. For instance, in one case, the pain was experienced on the side opposite the affected frontal sinus, which was owing to the fact that the latter extended beyond the midline in the form of a recess that failed to drain adequately through the main cavity. In another case the pain was located at the external apophysis of the frontal bone and radiated to the temporal region. Operation revealed an enormously extensive frontal sinus with a posterolateral recess that was almost completely shut off from the rest of the sinus by a nearly complete bony septum.

Réthi [177] describes the picture of trigeminal neuritis as distinguished from homonymous neuralgia and suggests the injection of 3 per cent solution of hydrogen peroxide (0.5 cc.) at the point of exit of the nerve, with passage of the needle into the canal for a distance of several millimeters. The resulting relief is prompt in true neuritis but has no effect in pure neuralgia.

Aschan's [178] monograph deals with the results of his experiments on rabbits, carried out for the purpose of explaining the phenomena of aero-otitis and aerosinusitis from the point of view of pathogenesis. He seeks to answer the following question: 1. Why are uniform clinical pictures caused by both an increase and a decrease of the ambient pressure? 2. Why do otoscopic changes appear so frequently without subjective symptoms? 3. Why does suppurative otitis media appear so rarely despite the frequent existence of rhinopharyngitis? 4. Why does the morbid condition persist for several weeks despite prompt therapeutic equalization of the air pressure?

As a result of his experiments it appears that the true cause of the pathologic changes observed is not the mechanical obstruction at the ostium or the tube, but rather the changes in oxygen pressure. The experiments included morphologic studies of the lungs and other organs. Histologically, the changes are characterized by edema, hyperemia, bleeding by diapedesis and transudation in the respective cavities. Apparently the changes observed were the same whether the oxygen content was diminished or increased. Oxygen poisoning, therefore, produces the same result as oxygen deficiency. The author states that "the experimentally observed changes in the present investigation should,

176. d'Ajutolo, F.: Alcune osservazioni sull'andamento e sulla sede del sintomo dolore nelle affezioni infiammatorie delle cavità paranasali, Riv. oto-neuro-oftal. **23**:200 (May-June) 1948.

177. Réthi, A.: Rhinogenous Trigeminal Neuritis and Its Therapy, Hungarian M. J. **46**:1901 (Nov. 16) 1947.

178. Aschan, G. K.: Aero-Otitis Media and Aerosinusitis, Acta oto-laryng., 1948, supp. 69, p. 9.

in the first place, be regarded as part phenomena in general physiological processes caused by oxygen deficiency and oxygen poisoning." This would "explain why different forms of 'barotrauma' produce identical clinical pictures, why, further, otoscopic changes may be found without any clinical symptoms and, finally why the risk of complication with suppurative inflammations is practically non-existent."

Dohlman's case [179] was of a woman aged 54 in whom the onset of the rhinorrhea was attributed to acute rhinitis. Careful examination revealed a flow from one side of the nose amounting to 10 cubic centimeters per half-hour. The fluid exhibited a low albumin content, 485 mg. of chlorine and 95 mg. of sugar per hundred cubic centimeters after centrifuging. Cystochrome injected into the subarachnoid space appeared in the discharge, apparently coming from the region of the middle turbinate. The patient was operated on via an external incision, as for a radical frontoethmoidectomy. The roof of the ethmoid and frontal sinuses was exposed, and it seemed that the fluid was coming from a narrow fissure in one of the ethmoid cells. This area was smoothed off and covered with a muscle flap and mucosa from the turbinal. However, the procedure failed to halt the flow. A second operation was performed. This time, when the previous operative site was reopened, it was observed that the fluid was coming through one of the olfactory perforations along a stalk of the nerve. The entire olfactory groove was opened up and the cribriform plate removed. The dura was elevated from the bottom of the olfactory groove and the crista galli, so that the flap could be overlapped and united. This was then covered with muscle and mucosa of the septum and turbinal and held in place with a tampon saturated with penicillin and sulfonamide. The spinal fluid was withdrawn twice daily via lumbar puncture. Dressings were removed in ten days. When examined six months later, the patient showed no evidence of further discharge.

Smith [180] gives a clear and sensible statement on the significance of headache in connection with sinus disease. It is a frequent symptom of sinus infection, but in some cases may bear no relation to that condition. On the other hand, headache may be caused by sinus disease in which the lesion is not discovered. He describes the typical frontal headache which comes on in the morning and disappears in the afternoon and is characteristic of frontal sinus disease. He points out that the occurrence of this symptom may be atypical in persons who work at night and sleep most of the day. The author describes sphenoid headache as being more

179. Dohlman, G.: Spontaneous Cerebrospinal Rhinorrhoea: Case Operated by Rhinologic Methods, Acta oto-laryng., 1948, supp. 67, p. 20.

180. Smith, A. B.: Symposium on Headache as Symptom: V. Headache from Sinus Infections, Edinburgh M. J. **55**:183 (March) 1948.

noticeable in the vertex and occiput; ethmoid headache causes pain between the eyes and in the temples. Sluder's lower half headache may be due to ethmosphenoid infection. Maxillary sinus disease, in the author's opinion, rarely causes headache. In this connection, he advises against attributing too much importance to slight or moderate cloudiness of the antrums when headache is a predominant symptom. No relation exists between the two in a large number of these cases, as many of them are of psychogenic or neurotic origin.

Vacuum headache is still a definite entity to be reckoned with and may be associated with conspicuous roentgenographic findings, as proved in the many cases of barotrauma reported after the recent war. In young adults or adolescents, headache may be due to the rapid expansion of the sinuses. The author makes this assumption by analogy with those cases in which severe mastoid pain has been observed to be caused by active underpneumatization during this period of life.

Vaheri [181] investigated the incidence of nasal infections in 1,294 cases of scarlet fever. Symptoms were present in 44.5 per cent of the group, the frequency being higher in children under the age of 5 years and in winter and spring than in summer. The average duration of nasal discharge was seventeen days, but in some instances it lasted as long as eight weeks. A six day course of treatment with penicillin shortened the duration of illness in most instances. Complications involving the accessory sinuses were diagnosed in 37 cases, or 2.9 per cent of the entire group, and responded to conservative treatment.

Bentzen [182] did some research on the matter of air-borne infections in the otorhinolaryngologic wards of a Danish hospital. Cultures were taken from all the patients, members of the resident staff, visitors, the air and the bed clothing, and the results were tabulated. Three hundred and twenty-five patients were examined for Pneumococcus cross infection, which was detected in 56. Of 659 patients examined for Streptococcus, 88 were noted to have a cross infection. It was shown that the bedding was responsible for the infection in many cases. Visitors also played a part, and the members of the resident staff were responsible only to a minor degree. Air-borne infection was a minor factor. The author believes that the incidence of cross infection is high enough to warrant limited isolation of all new patients for a period of observation, to determine whether they are carriers or not. Visitors should be strictly controlled and members of the resident staff routinely examined.

181. Vaheri, E.: Scarlatina Rhinitis and Its Complications, Acta oto-laryng., 1948, supp. 74, p. 165.

182. Bentzen, O.: Investigations into Infection by Air and Dust in an Ear, Nose and Throat Department, Acta oto-laryng. **36**:456, 1948.

Hájek [183] describes 3 cases of extrapulmonary tuberculous disease of the region of the nasal cavity, frontal sinus and lymph nodes. The first of the cases resisted the usual therapeutic measures for two years, and similarly the 2 other cases could not be influenced by the therapy employed. Finally, administration of high doses of calciferol (crystalline vitamin D_2) was followed by complete cessation of the specific processes. In the first 2 cases signs of an overdosage of vitamin D did not occur, but in the last case slight symptoms of intolerance appeared after three months' use of calciferol, which quickly disappeared on diminution of the daily dose.

Fischer and Fischer [184] report an interesting case of infection in the pterygopalatine fossa, causing necrosis of the orbital walls with serious disturbance of ocular function and extensive external swelling and pain. The orbital involvement was demonstrated on roentgenograms and, while some relief was obtained after exploration of the temporal fossa and evacuation of fluid, the improvement was only temporary. Following the suggestion of Sewell, the authors exposed the fossa by way of the antrum as in the Caldwell-Luc operation, removing the posterior wall and evacuating a large amount of muddy brown fluid. The opening was enlarged to approximately 2 cm. in diameter, some necrotic bone was removed and adequate drainage was provided into the nasal cavity. The recovery was complete.

25 East Washington Street.

183. Hájek, F.: Large Doses of Vitamin D in Therapy of Tuberculosis Otorhinolaryngologic Diseases, Časop. lék. česk. **87**:727 (June 11) 1949.

184. Fischer, N. A., and Fischer, A. J.: Necrosis Within the Pterygopalatine Fossa, Arch. Otolaryng. **48**:337 (Sept.) 1948.

Abstracts from Current Literature

Ear

A Statistical Study of Incapacitating Hearing Impairment in One Thousand Clinic Patients. Maurice Saltzman and Marguerite C. Eversden, Ann. Otol., Rhin. & Laryng. **58**:345 (June) 1949.

The authors, in processing 1,000 patients with deafness for rehabilitation by means of hearing aids, accumulated data forming an audiologic survey of the deafness problem in Philadelphia. No doubt similar findings would result from surveys in other industrial cities in the temperate zone.

Some hearing impairment was traceable to aural infections occurring before the days of chemotherapy. The acoustic trauma incidental to modern civilization and two great wars are additional factors, but in the majority of cases, hearing defects resulted from the natural wear and tear of life and hereditary predispositions. These groups include most cases of perceptive deafness, arteriosclerosis and otosclerosis. Institutional cases and total deafness problems were excluded from the study. Attention was directed to the person who could hear speech when loud enough and distinct enough but who sought help when his difficulty began to cause him embarrassment.

As prerequisite for classification, each patient was scrutinized on the basis of history, medical, neurologic and otoscopic findings. The audiologic study consisted of audiometry, fork tests, speech hearing tests and observation of the patient's speech and reactions to sound stimuli.

In 66.2 per cent of the cases was shown a defect in the perceptive mechanism, a condition most common in the older age groups. However, in children, deafness is most frequently of the perceptive type. Conduction deafness of a handicapping degree was encountered rather infrequently, occurring in only 1.8 per cent of the cases. Mixed deafness constituted 14 per cent in the series, while deafness due to neurologic and psychogenic causes occurred in 0.8 per cent and 0.3 per cent of the cases, respectively.

Otosclerosis was the etiologic factor in 16.9 per cent. There was a ratio of male to female of 1:3. The disease was found to produce a deafness problem rather infrequently before the third decade of life. Activation of an otosclerotic process during the menopause frequently left patients with a very severe hearing loss. In the series, 66 of the otosclerotic patients were 51 years of age or older, leaving 103 in the younger age group. Approximately one half of the latter group could not be considered suitable candidates for fenestration surgery owing to complicated audiologic pattern or medical or psychologic contraindications. In the opinion of the authors, the fenestration operation could have been offered conscientiously to only 50 of the 1,000 handicapped hard of hearing.

Steele, Kansas City, Kan.

Some Observations upon the Otological Effects of Streptomycin Intoxication. M. R. Dix, C. S. Hallpike and M. Spencer Harrison, Brain **72** (pt. 2):241, 1949.

This article deals with certain aspects of the central nervous mechanisms of labyrinthine and optokinetic nystagmus. As is well known, both types of nystagmus exhibit the same rhythmic alternation of slow and rapid eye movements. Further-

more the responses, if elicited together by means of simultaneous labyrinthine and retinal stimulation, exhibit well marked summation and cancellation effects.

These facts provide the basis for the opinion of J. Ohm that both responses must depend on a common mechanism in the central nervous sys.em which he identifies with the vestibular nuclei.

Now it would seem likely from the recent work of Winston and his co-workers of Philadelphia that the vestibular symptoms which occur in the course of streptomycin therapy are due to destructive changes in the vestibular nervous pathways including the vestibular nuclei. Accordingly, the validity of Ohm's theory was investigated by Dix, Hallpike and Hood in 12 subjects who had been treated with streptomycin in doses likely to induce vestibular intoxication.

The following results were obtained:

In 11 of the 12 subjects the caloric responses were abolished or considerably reduced.

In all 12, the galvanic responses were also abolished or considerably reduced.

Nevertheless, in all 12 the optokinetic responses were entirely normal.

The authors argue that if, as seems likely from the work of Winston and others, the reduction of the caloric and galvanic responses were due to destructive changes in the vestibular nuclei, then contrary to the views of Ohm the central nervous mechanism of optokinetic nystagmus must be entirely independent of these nuclei.

NOTE.—A previous abstract of this article, prepared by Dr. Benjamin H. Shuster, was published in the May issue of the ARCHIVES, page 764.

AUTHORS' ABSTRACT.

FENESTRATION OPERATION FOR OTOSCLEROSIS. Editorial, J. A. M. A. **140**:682 (June 25) 1949.

Since Dr. Julius Lempert first described the one stage method for surgical management of clinical otosclerosis, improvements in technic have been made. Means for ascertaining preoperatively the likelihood of obtaining practical and serviceable results have been improved.

For restoration of practical hearing it is important that the bone conduction for hearing in the 512, 1024 and 2048 pure tone frequencies be not lower than the 30 decibel level.

The decibel level of bone conduction hearing should be at least 25 to 30 decibels higher than the decibel level of air conduction hearing.

With an adequate cochlear nerve function reservoir, no matter how great the preoperative hearing loss, restoration of practical hearing is considered possible.

Dr. Lempert has described in detail his "bone-dust free fenestra nov-ovalis technic," the main principle of which is the prevention of the entrance of bone dust into the operative area, since the bone dust may be responsible for osteogenesis later. Osteogenesis interferes with or closes the fenestrum. The technic includes burnishing the fenestral rim with pure lead to prevent osteogenesis at the freshly cut bony edge.

Since the original operation was described, Dr. Lempert has performed 3,700 fenestration operations and 450 revisions.

The most frequent adverse complications have been osteogenesis at the operative site, bleeding, or oozing postoperatively, development of postoperative inflammation of the tympanomeatal flap and postoperative labyrinthitis. The last-mentioned three complications have been almost completely controlled by improvements in operative technic and the administration of 300,000 units of penicillin each day for ten postoperative days. The introduction of lead burnishing technic should greatly reduce the number of closures of the fenestra from osteogenesis.

Success depends on the proper selection of patient, operative technic used and the careful evaluation of preoperative and postoperative audiometric measurements.

Dr. Lempert estimates that unaided hearing can be restored by operation in approximately 80 of 100 patients considered most suitable for operation and that at the end of two years hearing regression will have occurred in 20 of this group of 80, thereby netting a 60 per cent successful two year result. Results at the end of two years are considered essentially permanent. With the latest advance better results are expected, but Dr. Lempert warns that there is great need for utmost caution in evaluating results in order that accurate preoperative prognoses may be made.

Other authors report a much higher percentage of successful operative results. Shambaugh and Juers (ARCH. OTOLARYNG. **43**:549-567, 1946) in recent studies using a modified technic in a group of patients designated as class A (ideal), a group which may be said to correspond to Dr. Lempert's (100 most suitable for operation), stated that successful two year results are possible in 80 per cent of the cases.

Shambaugh's technic includes enchondralization of the fistula, placing the fistula on the horizontal semicircular canal laterally and the use of a sea sponge and head frame technic to prevent postoperative serous labyrinthitis.

In 1946 Shambaugh stated that 92 per cent of the patients of an operative series measured audiometrically two years postoperatively showed improvement, although not all patients had maintained their maximum gain.

As standards of selection of patients, operative technic and methods of postoperative and preoperative audiometric evaluation have changed this estimate of prognosis has apparently been revised downward.

Other authorities suggest that the 20 per cent difference in the estimate of good prognosis (60 per cent by Lempert and 80 per cent by Shambaugh) probably does not relate to a real difference in operative results obtained or technics employed but is a factor of difference in standards of patient selection and differences in concept of what may be considered a successful result. GORDON, Philadelphia.

Larynx

CONTACT ULCER GRANULOMA. GORDON B. NEW and KENNETH D. DEVINE, Ann. Otol., Rhin. & Laryng. **58**:548 (June) 1949.

Vocal abuse is the chief etiologic factor in the production of contact ulcer granuloma, although it would appear that the trauma or pressure associated with tracheal intubation for general anesthesia is of growing significance. Symptoms are discomfort in the larynx, hoarseness or pain extending to the ear and sometimes hemoptysis and chronic cough. The disease most frequently occurs in physicians, farmers, managers, ministers, agents and salesmen.

Treatment is by surgical excision with intravenous thiopental sodium (pentothal sodium®) supplemented by curare (d-tubocurarine chloride), utilizing suspension laryngoscopy for exposure of the lesion. Cup forceps are used to remove the granuloma and the base of the wound is then touched with surgical diathermy.

The patient is sent home after a few days on a regimen of silence. Reexamination is made in one month. If the granuloma has not recurred or is smaller than before, periodic observations are continued. In the presence of recurrence, removal is repeated, but at least one month or longer should elapse between operations. The importance of subsequent vocal reeducation following excision and diathermy is stressed.

The authors have noted that patients with symptoms for months or years rather than weeks will require less frequent removal. In addition, if the granuloma bleeds readily when touched or is soft and friable when grasped, there is a poorer chance that the patient will be well after only one operation than if the granuloma is hard and fibrosed.

Fifty-three patients with contact ulcer granuloma were examined. Of these, 9 presented a history of tracheal intubation prior to the onset of their symptoms. Contact ulcer granuloma which followed tracheal intubation occurred oftener in women than in men, the ratio being 2:1, while in the group of 44 patients with no history of previous tracheal intubation no females were afflicted. Contact ulcer following tracheal intubation is usually bilateral, and development apparently is not related to the length or the kind of operation, route or difficulty of introduction of the intratracheal tube. STEELE, Kansas City, Kan.

STUDY OF THIRTY CASES OF FOREIGN BODY IN THE UPPER AIR PASSAGES AND UPPER DIGESTIVE TRACT. OSVALDO EMHART, Rev. otorrhinolaring. **9**:3 (March) 1949.

Emhart presents 30 cases of foreign bodies in the airways and the upper digestive tract. The material covers a large variety of foreign bodies, 17 of which were lodged in the airways and 13 in either the hypopharynx or the esophagus. The age group of his patients ranged from 1 year and 4 months to 12 years. Most commonly the foreign bodies were present from one to three days. While some of the endoscopies were done with general anesthesia, most of the patients did not require any anesthetic. Three patients required a tracheotomy. Three patients died. In 1 case of death the endoscopy was long and presented a difficult extraction of a watermelon seed in the bronchus; the second death was caused by heart failure, and the third by secondary pulmonary complication. The author does not use any sedation prior to endoscopy. In complicated cases he advocates the use of both sulfonamide drugs and penicillin. In conclusion, he advocates the establishment of bronchoesophagoscopic centers in which their personnel have adequate specialized training. PERSKY, Philadelphia.

ENDOBRONCHIAL TREATMENT OF PULMONARY ABSCESS. JENS L. HANSEN and CARSTEN M. SMIDT, Acta oto-laryng. **36**:505 (Sept.-Dec.) 1948.

The authors state that all patients with symptoms that suggest pulmonary abscess should receive bronchoscopy. It is important that this procedure be carried out at an early stage. If the lesion is not cancerous and is a bronchial stenosis or an occlusion, the procedure may often be curative. If the procedure is not carried out at an early stage there may be permanent changes that cannot be corrected.

If there is no lesion of the bronchi per se, which is the usual situation in most instances of pulmonary abscess, the aspiration of the pus through the bronchoscope may prove beneficial. It is usually the case in these instances that the pus can be aspirated only from the bronchus adjacent to the abscess and not from the abscess itself. The process of coughing into the bronchus during the procedure empties the abscess into the bronchus. The authors report 4 cases. In the fourth is discussed a new method of direct bronchoscopic drainage from the abscess and instillation of penicillin. ROBERT LEWY, Chicago.

Nose

PENICILLIN AEROSOL THERAPY IN SINUSITIS. FRANK J. HYNES, Ann. Otol., Rhin. & Laryng. **58**:189 (March) 1949.

The author describes the technic of penicillin aerosol therapy and presents reports of cases. Of 212 patients treated, 58 were classified as having been cured clinically; 34, with marked improvement; 45, with moderate improvement; 19, with slight and 34 with no improvement. The bacteriologic studies confirmed those of other investigators in that, for the most part, patients continued to show the same type of bacteria before, during and after treatment, regardless of the clinical results obtained. The author believes that in properly selected cases penicillin aerosol with oxygen is a valuable aid in treatment. He states that aeration of the respiratory mucosa is important in treatment of infections of the area, and this procedure, of course, is aided by the oxygen. Oxygen alone may inhibit the growth of secondary invaders. Penicillin aerosol with oxygen, he maintains, is the treatment of choice, provided that empyema of the sinus is not present. If it is present the empyema must be drained. If the organism is sensitive to penicillin, penicillin aerosol may shorten the duration of treatment.

In his conclusion Hynes makes the following statements: 1. In allergic rhinitis the treatment is contraindicated except in proved cases of bacterial allergy or unless there is complication by infection of the sinuses. 2. In the common cold results obtained are due purely to the local and general care plus the use of oxygen. 3. In acute sinusitis the results are entirely dependent on the presence or absence of inspissated pus in any of the sinus cavities or in a chronic bronchiectatic cavity. 4. With sinusitis in children spectacular results are usualy shown with penicillin aerosol together with proper local treatment. Persistent sinusitis calls for repeated cultures. Autogenous or other vaccine therapy and the administration of streptomycin may be necessary. 5. Acute exacerbations of chronic sinusitis may be controlled by aerosol therapy, and when this therapy is accompanied with use of oxygen chronic infection often subsides. Autogenous vaccines may be helpful. 6. With chronic sinusitis spectacular results may occasionally be shown, although patients with this condition are advised that they should expect no relief except for acute exacerbations. 7. In cases of acute laryngitis and acute bronchitis treatment was made with aerosol and oxygen, and in almost every case spectacular relief was gained. 8. A number of obviously psychoneurotic patients were treated, but with entirely negative results.

MILLER Philadelphia.

PHYSIOLOGY AND CLINICAL STUDIES OF THE SINUSES. EUGENIO ROMERO DIAZ, University of Cordoba, Argentina, 1949.

In an extensive thesis Diaz presents a complete survey of the nasal accessory sinuses. He discusses the anatomy, histopathology and physiology. He makes an extensive discussion of the functions of the nose per se and of its integral parts composed of the turbinate bodies and the paranasal sinuses. The physiology of the mucosa of the sinuses and the action of drugs on the cilia and the nasal mucosa are described in great detail.

Many problems are discussed from the standpoint of focal infection and the relation of the sinuses to the respiratory tract, such as pneumonia, asthma and bronchiectasis. There is an extensive discussion under diagnosis and symptomatology and of conditions commonly associated with sinus infection and the complications which may arise. Under the subject of treatment, antral punctures

and sphenoidal irrigations only are mentioned; surgery is not discussed. The entire thesis is replete with illustrative cases emphasizing the various points of the discussion.

PERSKY, Philadelphia.

Pharynx

ENDOCRANIAL HYPERTENSION OF RHINOGENOUS ORIGIN. C. E. PIERANGELI and A. SARTORI, Oto-rino-laring. ital. **15**:270, 1947.

The authors, after a review of the literature on the rhinogenous hypertensive endocranial syndromes and on their genesis, present a case of serous meningitis of acute recurrent character, secondary to congestive bouts of infection of the pharyngeal tonsil.

After every attack of catarrhal pharyngotonsillitis, the patient would be affected with frontal and occipital headache, spinal pain, constipation, vomiting and drowsiness. During one of the attacks a spinal tap was performed, and clear fluid under considerable pressure was obtained, biochemically and cytologically negative. Adenoidectomy caused the rapid and complete subsidence of all the symptoms.

MELUCCI, Philadelphia.

THE ROLE OF INTRAORAL PRESSURE IN THE PATHOLOGY OF THE TONSIL. G. REVESZ, Pract. oto-rhino-laryng. **11**:193, 1949.

Tonsillar infections can be exhibited either by generalized symptoms on the basis of focal infection or by local manifestations. Revesz has shown by a series of pressure experience and the use of a manometer that changes in air pressure in the mouth and in the pharynx on the tonsils can produce absorption of the infectious material. In summarizing his experiments he makes the following statements.

1. That because of their position in the faucial pillars the tonsils are very apt to become an infective focus.

2. The action of the muscles of the faucial pillars exercises a considerable amount of force on the tonsils during deglutition.

3. The pressure of these muscles exerted on the tonsils is approximately 0.6 Kg., the equivalent of the pressure tension 0.45 Kg. per square centimeter in the center of the tonsillar tissue.

4. During deglutition a pressure of at least 130 mm. mercury develops in the oral cavity, equivalent to a tension pressure of 0.22 Kg. to the surface of the tonsil. With this pressure added to the tension pressure of the muscle, the surface of the tonsil is exposed to a pressure of 0.8 Kg. during the act of deglutition.

PERSKY, Philadelphia.

Miscellaneous

AUREOMYCIN. N. V. PETERSON and R. B. HADLEY, Eye, Ear, Nose and Throat Monthly **28**:422 (Sept.) 1949.

The authors present a series of 18 cases of chronic and acute sinus infections in which the use of aureomycin proved effective. There is a wide range of bacterial effectiveness for both gram-positive and gram-negative organisms. Advantages include the lack of toxic reactions as well as the ease of administration, comparatively small dosages and lack of sensitivity reactions. The side effects appear to be negligible.

JENNES, Waterbury, Conn.

Society Transactions

SECOND INTERNATIONAL AUDIOLOGY CONFERENCE

Prof. Gunnar Holmgren, President
London, England, July 15 and 16, 1949

Informal Report. Dr. Norton Canfield, New Haven, Conn.

The Conference was called to order by the president, Prof. Gunnar Holmgren, of Stockholm, Sweden. The opening remarks were made by Mr. Scott Stevenson, of London, England, president of the National Institute for the Deaf, who welcomed the guests from about 20 countries.

President Holmgren then reviewed the meeting of September 1948, held in Stockholm (complete transactions in *Acta Otolaryngologica*, Supplement 76), and mentioned that the International Audiology Conference was taking on a more definite organizational character, the details of which would be reported later at this meeting. The field of audiology is not new, but the name is, and the specialty includes many professional people. Organization of the Conference is complicated and delicate, but gradually the Conference will assume its rightful place in audiologic thought.

Secretary Huizinga, of Gronigen, Netherlands, pointed out that the difficulties at present were chiefly two: the need for financial assistance to carry on the meetings and to publish the transactions and the need for an office from which information could be disseminated to the various countries interested. He made three proposals: (1) that there be a permanent two member committee from each country; (2) that the central committee should be known as the Council, and (3) that the Council invite specially qualified visitors to the meetings in the future to supplement the delegates from each country.

Prof. Maurice Sourdille, of Strasbourg, France, then read a paper making a plea to organize an international society of otorhinolaryngology and to abandon the term audiology because there are already too many branches of the main specialty, such as phoniatry, bronchoesophagology, maxillofacial surgery and otoneuro-ophthalmology.

To this, Dr. Canfield, of New Haven, Conn., replied that audiology was firmly fixed in the language, that the term originated with Willard B. Hargrave, instructor in audiology, Claremont College, Los Angeles, and that it is now in the medical dictionary at the suggestion of the Committee on the Conservation of Hearing, of the American Academy of Ophthalmology and Otolaryngology. The term audiology cannot be abandoned because much of the activity of organizations in the United States for impaired hearing was built around the term and because at least two universities now have professors of audiology.

Secretary Huizinga supported the use of the term audiology and indicated that it should be used by properly qualified professional people.

Dr. Grabbe, of Brussels, Belgium, agreed that the organization of the Conference should go on as directed by the Council and mentioned the necessity of support from the existing societies in each country.

Dr. Groen, of the Netherlands, asked how persons could become members of the Conference and whether they needed to be otologists as a requirement for membership.

Dr. Canfield replied that final organizational plans would be stated by the Secretary and that representatives from a country need not be otologists. Physicists, psychiatrists, speech experts were all welcomed. The only qualification was that applicants should be representative audiologists, as that term was defined at the meeting in September 1948.

Dr. Ferreri, of Rome, Italy, proposed standardization of audiometric technics and requested criteria for patients who could benefit by the fenestration operation.

Dr. C. Kinney, of Cleveland, commended the Council for its activities during the past year and suggested that the organization of the Conference be not too specific and definite but rather be maintained as a meeting which will be informal and one where individual discussion will be encouraged. The important thing was to spend time on the problems of hearing.

Dr. Halliday, of Sydney, Australia, supported the remarks of Dr. Kinney and mentioned his recent visit to the United States. Committees of the Conference should be selected by men the caliber of Professor Holmgren.

Mr. Stevenson mentioned an important point. He had declined a request by manufacturers of hearing aids in England to attend the meeting, and he asked for an expression of opinion by the Conference about this subject in the future. In Britain, the manufacturers often gave useful information, but their "research" was never published. The National Institute for the Deaf had set standards for commercial hearing aids in an attempt to protect the public. Dr. Stevenson requested the Conference to bar the manufacturers and dealers from its meetings.

Dr. Hennebert, of Brussels, discussed the problem concerning hearing aid dealers and stated that the subject was a delicate one which would require different handling in each country.

Dr. M. Arslan, of Padova, Italy, proposed inviting a physicist or physiologist to the next meeting, and he was reminded that Harvey Fletcher, of New York, was present at this meeting and would present a paper later.

Dr. E. P. Fowler Jr., of New York, agreed that each country should select its own delegates and may include otologists, physicists, physiologists or any one really interested.

Dr. Bocca, of Milan, Italy, referred to Dr. Arslan's suggestion but added that public health officials and other administrators in each country might select the delegates.

Dr. Canfield stated that in the United States some of the representatives of the hearing aid manufacturers would be most welcome to professional meetings and that it did not seem right for the International Audiology Conference to accept Dr. Stevenson's suggestion to bar them. The question was referred to the Council without any unified expression of opinion on the part of the members of the Conference.

Friday Afternoon Session, July 15

President Holmgren announced that the scientific program would begin.

Dr. E. P. Fowler Jr. presented the first paper, entitled "Report of the Standardization Subcommittee," by Prof. E. Lüscher, of Basel, Switzerland, and himself. In summary, it was recommended that (1) standards already generally accepted by organizations, such as the American Standards Association or the International Standard Organization, be adopted by the Audiology Conference; (2) standards about which there is little controversy should be adopted, but for those who disagreed little harm would be done, and (3) standards about which there is still marked disagreement, such as calibration of audiometers, should be circulated to interested parties for their comment to help decide the final standards to be adopted.

Dr. Harvey Fletcher, of New York, mentioned first that he had spent his entire professional life as a member of a commercial laboratory which provided him with facilities for research that no university could provide. He was a member of scientific societies in the United States and closely associated with governmental programs for the deaf and hard of hearing. In his prepared paper, he developed the thesis that the hearing threshold for speech can be accurately determined from the pure tone audiogram. This speech threshold is the average of the two lowest thresholds in the pure tone audiogram taken from the readings at 500, 1000 and 2000 cycles per second re .0002 dynes sq. cm. Exceptions occur only when the pure tone audiogram curve is very unusual.

Dr. Kinney suggested that the individual authors send reprints of their papers to the members of the Conference as a means of dissemination of information.

Dr. Stevenson praised the research of Dr. Fletcher and said his previous remarks did not apply to manufacturers in the United States or to scientists working in commercial laboratories.

Dr. Fletcher replied that the point he wanted to stress was that there were good scientists in commercial groups in the United States.

Dr. E. P. Fowler Sr., of New York, discussing Dr. Fletcher's paper, stated that clinically Fletcher's method would not be so simple as presented because the U-shaped audiogram curves were difficult, speech tests varied in different languages and that adults differed from children in their response to any test.

Dr. E. P. Fowler Jr., in replying to Fletcher, stated that he had had the opportunity to read the paper before the meeting and that results of audiometry depend on the speech tests used. Some word tests only test low frequencies. In Dr. Fletcher's audiograms there were very few of the sharp drop type. Dr. Fowler mentioned that many present day speech tests are not accurate and that they do not necessarily indicate the true ability to hear speech.

Secretary Huizinga, in discussing Dr. Fletcher's paper, asked whether audiograms were made in silence and what experience with different dialects indicate.

Dr. Frye, of London, stated that understanding speech was different from hearing speech. He suggested accepting speech tests now because they have the variability that occurs in the everyday life of those who hear.

Dr. Bocca mentioned his own research on speech testing with the use of newspaper copy in an ordinary room. He obtained good correlation in 300 patients with conduction deafness.

Dr. Fletcher stated that the method he proposed was as simple and as accurate as any presently used speech method for obtaining the speech threshold. If the real loss for speech is desired, a much more involved and complicated test is necessary. Further work is being done in complicated cases. Simplicity is the keynote of the method he suggests, and so far no other test is any better.

Dr. Lennart Holmgren, of Stockholm, reported on the activities of the Swedish Royal National Committee during the year under the subjects of fenestration surgery, noise and prevention of acoustic trauma and hearing aids.

Cases for fenestration should be controlled by the Hard of Hearing Institute and not by otologic clinics of universities. Not more than 10 per cent of all those who need audiologic help are patients requiring fenestration.

Managers of industry and members of the Armed Services should know of the hazards of noise. Employees in industries with noise should be instructed to avoid trauma, and researchers should be encouraged to provide better ear defenders.

In the field of hearing aids a decision was being approached. Should the British or American way be adopted? Sweden would probably go the middle way, with some governmental regulation of private business. Competition was very impor-

tant, and the price question would solve itself. Hearing aids should be sold only through established offices; the buyer should be able to return it in one month if it proves unsatisfactory. Aids should be easily available only to those who can get good results; the remaining hard of hearing people (25 per cent of all) need special attention from audiology centers. There should be some control of advertising, and only correct statements should be disseminated to the public.

Dr. Canfield then told of the activities of the Council of the Audiology Conference during the past year in an attempt to associate the present group with one or more of the already existing international health and education organizations. He read excerpts from a letter from the World Health Organization, which is part of the United Nations, and from the World Medical Association, which is now representative of most of the medical associations in the world. Contact with UNESCO, also part of the United Nations, has not been fruitful as yet, but efforts are continuing. The World Medical Association has requested the International Audiology Conference to send observers to its next meeting to be held in London this autumn. Dr. Charles S. Hallpike and Dr. T. Cawthorne, of London, were designated by President Holmgren for this assignment.

Dr. J. Bijtel, of Amsterdam, told of the sound progress being made in the Netherlands. There are institutes for the deaf in Amsterdam, Rotterdam and The Hague. The government has much to do for the partially deaf, and opportunities for their help are not yet well organized.

Dr. Callicetti, of France, stated that the "Institute National Surdité" is to be established and will be the center of activity in his country.

Dr. Hennebert spoke of the distribution of hearing aids in Belgium and the difficulties with control of apparatus.

Dr. J. M. Tato, of Buenos Aires, Argentina, described methods of selecting frequencies for speech analysis and presented the organizational plans for the "Institute d'Audiolgie d'Argentine," which is now before their legislature for final adoption.

Dr. Vetter, of St. Gallen, Switzerland, gave an account of activities in Switzerland during the year, including progress in labiology, formation of a central office of information, the publication of a periodical and the confederation of 52 societies. Switzerland may not be large enough for large audiology centers, but progress is being made there nevertheless.

Saturday Morning Session, July 16

The papers on this day were presented entirely by the English members who had the responsibility for operating their government's schemes for rehabilitation of the deafened, under the authority of the National Health Act.

The first paper was read by Sir Wilson Jameson, Chief Medical Officer, Ministry of Health. He stated that in 1941 the government started rehabilitation of all Armed Service men, including the deafened. There had been good cooperation from the Medical Research Council, and it became obvious that a special research unit was necessary for the attention to the deafened. In 1945 the unit was organized and three committees were appointed which concerned themselves, respectively, with medical and surgical problems; educational treatment, and electroacoustics. In 1945 the committee on electroacoustics reported recommending specifications for hearing aids and audiometers.

Mr. L. Wilkins, Senior Research Officer of the Social Survey of Deafness, reported in his social survey on samplings from random questionnaires of 31,000 people over 16 years of age. From this material it was concluded that 3,000,000 hearing aids would be applied for as soon as government aid was available. To date,

25,000 aids have been issued and work is continuing with staff training for issuing more. Other services to the deaf are being considered, and in time the government will be able to develop satisfactory service for the deaf.

Dr. Hallpike stated that health authorities had previously neglected deafness. As liaison officer for the Medical Research Council attached to all three of the aforementioned committees, he outlined in more detail what each committee was doing and the recommendations already made.

Mr. W. G. Radley, Controller of Research, National Physical Laboratory, stated that the first objective was to recommend specifications and develop a satisfactory hearing aid. The maximum intelligibility for speech was the prime requirement of the aid, and use for listeners of music was secondary. To this end special attention was given to the energy spectrum of the aids, the various frequencies to be transmitted and the character of the hearing of the listener. A master hearing aid was used, and in the practical tests many psychologic difficulties were encountered. For most of the tests recorded speech of 50 single words (25 from a male and 25 from a female voice) was used. Sixty-three patients were used for the tests from the Education of the Deaf Institute in Manchester, and 165 patients from among the deafened at the National Hospital, Queens Square, London. The pure tone losses of these 228 patients ranged from slight to an average of 90 decibels above threshold. A statistical examination of the results obtained showed that enough patients had been tested for reliable information. The final conclusion was that an aid that would give an output which up to 750 cycles would increase at the rate of 12 decibels per octave—from 750 cycles to 4000—would either be constant or increase gradually up to 12 decibels at 4000 cycles; and above 4000 cycles no frequencies are needed.

The next stage was to construct an aid at a reasonable price to meet the requirements. Two aids were made, one with a flat external receiver and one with an insert receiver. The aid compared favorably with an American aid made in 1944. In 1948 and 1949 there were 42,500 Medresco Mark I aids manufactured.

Mr. R. S. Dadson, of the Physics Division of the National Physical Laboratory, told of the work under his direction. At first many tuning forks were calibrated, and then the calibration of several commercial audiometers was investigated. These instruments showed gross discrepancies in actual testing, so output specifications were drawn up. Bone conduction testing is still being investigated.

Mr. T. S. Little, of the Department of Education of the Deaf, of Manchester University, told of experiences with group audiometry for children. He stated that no deaf child should be undetected but that screening should not put those with adequate hearing into schools for the deaf. The picture test of the Medical Research Council was described and the need for controlled amplification of speech indicated. The mental age was mentioned as of great importance in testing small children. Concerning the use of hearing aids by children, it was stressed that they must be used in conditions of ambient noise comparable to normal hearing situations and that characteristics of the room in which aids are used should be part of the specifications.

Mr. Michael Reed, Administrative Officer of the Ministry of Health, told of the responsibility of the Minister to supply hearing aids "without charge," to maintain them and to supply the necessary batteries. The two problems were production and distribution. An estimate of 150,000 was used as the number of persons who would or could accept a government aid. This figure was arrived at before the social survey of Mr. Wilkins. On the first contract 75,000 Medresco Mark I aids were required. Some components came from the United States at first, but all are now made in the United Kingdom. Some were available on July 5, 1948

when the Health Act was first in operation. There were few good clinics through which aids could be issued, and it was soon realized that the trained personnel to instruct patients was lacking.

The program for the deafened was provided at existing diagnostic clinics, of which there were 120 in the United Kingdom. At first there were only four clinics that were prepared to issue aids. Now 25 clinics issue aids, and soon there will be 10 more.

The training of clinic staff was first done in a two weeks' course. This was too short, so it has now been lengthened to six to seven weeks, which still may not be long enough.

Criticisms of the program have been many. Delay in getting aids has resulted in a waiting list of 60,000 in May 1949. There are many indigent members on this list, but pressure to supply commercial aids or even part payment for such aids has been completely resisted by the Minister of Health. So far only air conduction receivers have been issued. Bone conduction receivers are not available. About 25,000 aids have been issued, and the future of the program is now somewhat clearer.

Mr. E. A. Underwood, from the Ministry of Education, stated that by law education systems must include a school health service. The schools must discover and diagnose hearing loss, but treatment must also be carried out in many instances by the School Health Program. By the group audiometer tests before the war between 5 and 6 per cent of school children were found to have a loss in one or both ears of 9 decibels or more. By spoken voice tests used during the war only 0.5 per cent were detected, showing that about 80 per cent of those who were detected by audiometer were missed by voice testing. Surveys must be followed by adequate comprehensive special treatment, or the discovery of the cases is of no value.

The profoundly deaf in Great Britain are also under care of the Ministry of Education. There are 3,700 accommodations for such children, and 200 more are needed. Some of the problms with group hearing aids were discussed.

Miss Edith Wetnall, Director of Deafness Clinic, Royal National Throat, Nose and Ear Hospital, read a fine paper on the workings of one of the London clinics issuing the aids. The first aids delivered by the government were slow in coming, and when they did arrive, 50 to 60 per cent of the cords were broken, the professional staff was very much overworked and the technicians were too few. It was soon found that to hand out aids without adequate instruction and training of the patients was useless.

Three visits were required of each patient to do even a fair job. Fourteen aids could be issued per day, or about 40 per week. The technician who occupies the central position on the issuing team should be trained for 6 to 12 months; 4,222 aids have been issud in 12 months, and there is a closed waiting list of 4,000, with many more who are not yet on the list. In her concluding remarks, Miss Wetnall stated that the Medresco Mark II aid was good, but the batteries were heavy, the cords troublesome and no bone conduction receiver was available. There were many grateful patients.

Mr. Scott Stevenson, Director of the Deafness Clinic of the Metropolitan Ear, Nose and Throat Hospital, stated that they had been selecting commercial hearing aids since 1937, as a part of the program of the National Institute for the Deaf. Between 1937 and July 5, 1948, many free aids were dispensed.

Experiences with the Medresco Mark I Aid were then related. Of the first 600 hearing aids, 30 per cent were "duds" and would not function, but since then very few others were. The new Mark II aid is much better. Repairs and spare

parts were short. Of the first 7,000 aids issued 2,645 were to replace poor ones; 1,591 were repaired by taking parts from other aids. There were on the hospital list 4,000 priority cases, and the aged and those not requiring aids to keep or get jobs constituted 4,000 more. The battery consumption is about 1 B battery to 4 A batteries every eight weeks. The ear pieces are not standardized yet; 2 per cent need bone conduction aids, but none are available. From July 1948 to July 1949, 7,100 aids were issued. A course of staff training requiring six months to complete is in operation.

Mr. Reed stated that the government program included the following considerations for the future.

1. All patients will have individually molded ear pieces.
2. Aids will be of different colors.
3. Smaller batteries will be available.
4. A Medresco Mark III Monopak instrument is now in the research stage.
5. Distribution will include traveling clinics to 120 centers.
6. Future needs are being determined, and life of the present instruments is being awaited with interest.
7. Annual incidence of new cases is to be determined by survey.

Mr. Radley stated that 100,000 Mark II aids are being made now, but new designs will include automatic volume controls; substitutes for potassium sodium tartrate (Rochelle salt) in receivers, and new valves (tubes) with less battery drain.

Mr. Radley's talk concluded the presentation given by the Ministry of Health Officials. President Holmgren then stated that the morning had been very interesting and the procedure might serve as an example for future meetings in various countries. He thanked the speakers for their contributions and announced that there would be an exhibition of aids and audiometers made to government specifications after the meeting. Discussion of the papers presented then ensued.

Dr. Tato asked about the importance of selecting the best of several aids for each individual patient.

Dr. E. Fowler Jr. mentioned that pure tone testing for school children was very important in order to detect those children with a high tone loss. This pure tone testing was being done by the sweep technic and was found excellent.

Dr. Fletcher stated that he still held the view that a hearing aid output should match the audiometric loss in speech frequencies if there was not more than a 25 decibel difference in the frequencies up to 4,000. Any loss betwen 30 to 60 decibels in speech frequencies can benefit from a straight line response curve aid, but if an audiogram goes beyond this limit the response curve should incline one third as fast as the audiogram curve declines.

A plea was made for standardization of couplers so that the United States and Britain could be mutually more helpful. The group hearing aid with cut offs and compressors, so that the instructor could walk around the room and still deliver equal amplitudes to patients, was mentioned as a great help.

Dr. Kinney stated that laboratory work on hearing was most necessary, but that the otologist must "live with the patient" and get the complaints which cannot be overcome in the laboratory. Furnishing an aid rather than fitting one seemed to be the British policy. Referring to the issuing of aids as being without charge, as mentioned by Reed, was objected to because the National Health Service was being paid for by the very patients who were getting the aids.

Mr. Radley, in replying to Dr. Fletcher, said that when deafness was not extreme a mirror image of the audiogram for the output of the aid may be all right, but that speech intelligibility was more important.

Mr. Dodson said that relations of objective to subjective testing of receivers was being done with hearing aids, as with telephone receivers. An artificial ear was being used. Cooperation with the United States on the question of couplers could be arranged.

Dr. A. Glorig, of Washington, D. C., questioned the consideration of transient responses and asked who should and who should not be tried for the issue of an aid and how the audiometers were calibrated. Was the cadmium-nickel battery being tried? He stated that the British system, as outlined, put too much emphasis on the aid itself and not enough on the patient. He emphasized the need for training the patient in the use of the instrument, the importance of lip reading instruction and the value of a spare aid.

Afternoon Session, July 16

Mr. T. Cawthorne, secretary of Medical Research Council's Medical and Surgical Committee on Deafness, described the working of his committee in discovering the incidence of deafness. The survey of Mr. Wilkins (see below) was initiated, as was the Factory Otology Scheme, to determine the incidence of ear disease in civil life. Five factories were selected with a worker population of 57,000, of which 5.9 per cent had active ear infection and 14.6 per cent had had previous infections of the ears. With proper care, 80 per cent of these could obtain temporary cure of the infection. Daily attention to each patient was the most effective treatment, but this is impractical except when time away from work can be avoided. It was found that pain interfered most with the workers' efficiency and the resulting deafness interfered most with promotion.

As a result of the information obtained in this pilot study, the Ministry of Health was advised to include this type of ear disease treatment in the National Health Act program. The Factory Otology Scheme also made the deafness investigation much easier, since the treatment scheme was so beneficial there was no objection on the part of the workers or the employers to investigating the problem of noise trauma. Noise levels of 100 to 135 phons were encountered, and these levels are definitely hazardous.

Mr. Wilkins reported on "The Prevalence of Deafness in England, Scotland and Wales." He was asked by the Minister of Health to estimate the number of hearing aids likely to be required. In a 10 months' period in 1947 all persons interviewed were self-classified according to their hearing ability. The system of classification was similar to that adopted by Beazley of the United States Public Health Service Survey, but hearing tests were not used in the British survey, as they were in Beazley's system. Some conclusions are interesting.

Less than 5 per cent of the total number of people with hearing impairment were using hearing aids, 3 per cent in the lowest income groups and 12 per cent in the highest income groups. The deafened population was not generally disposed toward the use of aids: 11 per cent had no objections; 34 per cent were neutral; 41 per cent had minor reasons against its use; 17 per cent heard all they wished to hear, and 7 per cent had strong reasons against the use of aids. The first two groups, or 45 per cent, were then believed to constitute those who would presumably apply for governmental aid, a total in the population of 300,000. This figure was given to the Minister of Health as compared to that of 150,000, which was the number arrived at without the survey. Another interesting point was that only one half of the deafened population had ever sought medical attention.

Discussion of these two papers followed.

Secretary Huizinga spoke of his visit to Birmingham factories with Mr. Cawthorne, the experience of intense discomfort in a noise field of 100 and more

decibels and his relief when the Rüedi ear defenders were put on. He asked Mr. Cawthorne if the recently reported Russian experience that infection of the ears protects against noise trauma was found in the Factory Otology Scheme.

Dr. Tato asked about the prevalence of hearing loss in children and reported that it was 14 per cent in Argentina.

Dr. Lennart Holmgren, in replying to Secretary Huizinga, stated that slight chronic otitis media made the ear more sensitive to nerve deafness from noise and that the normal ear had a protective mechanism for loud noise which infection interfered with.

Dr. L. Rüedi, of Zurich, Switzerland, stated that the frequency range causing acoustic trauma was below 3,000 cycles. The Russians were incorrect about infection protecting the ear. For noise levels of 100 to 115 decibels above threshold, it was necessary now to select the workers who can tolerate it.

Dr. I. Hirsch, of Cambridge, Mass., said the question of noise deafness should be approached from the standpoint of preventing the noise from reaching the ear. Maximum insulation at the ear is about 50 decibels, so noise is better controlled at the source. Even 70 to 80 decibels of noise may injure some ears if they are exposed long enough. He mentioned the Bibliography of Hearing, now in preparation at the Psycho-Acoustical Laboratory at Harvard, in which already 4,000 entries are listed. In these entries, safe levels range in different articles from 60 to 140 decibels above threshold.

Mr. Johnston, of England, stated that some workers could hear through extreme noise and thought this must be a matter of training.

Mr. Cawthorne said that ear disease is not a protection against trauma from noise and that the two important factors were the intensity and duration of the noise. Workers are willing to protect their clothes but not their bodies.

Mr. Wilkins, in closing, said that the answer to the question of how many aids are necessary depends on the definition of "necessary." Is it a psychologic, sociologic or medical answer which is wanted? His estimate of 300,000 aids for England, Scotland and Wales was a minimum.

President Holmgren thanked the authors and discussers for their presentations, and the Second International Audiology Conference was adjourned.

Official proceedings of this meeting are to be published as a supplement to the *Acta Otolaryngologica*, in a forthcoming issue.

Book Reviews

If You Have a Deaf Child. Pp. 130. Published for the Illinois Annual School for Mothers of Deaf Children by the University of Illinois Press, Administration Building, Urbana, Ill., 1949.

This booklet, written for mothers of deaf children, is a major contribution to the otologist and to the educators of the deaf. At the University of Pennsylvania Hearing Center it is being given to every mother who has a child with a significant hearing loss.

The booklet is composed of chapters by various authors, including Mrs. Spencer Tracy, Dr. Francis L. Lederer, Dr. Harold Westlake, Dr. Maurice V. Moriarty, Dr. Jean Utley and many others. It covers the majority of problems present in the family of a deaf child. It is written in a very simple, lucid and interesting manner. Almost all the questions a physician is generally asked by a mother with a deaf child are answered in this booklet. Such problems as training deaf children and handling their emotional problems are very clearly explained. The use of hearing aids by children and how they work and the education of the deaf child are extremely well clarified. This booklet should be in the hands of every family in which there is a hard of hearing child.

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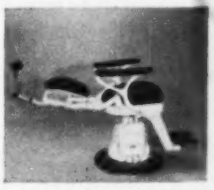

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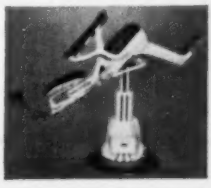

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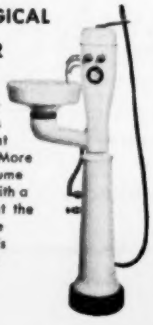

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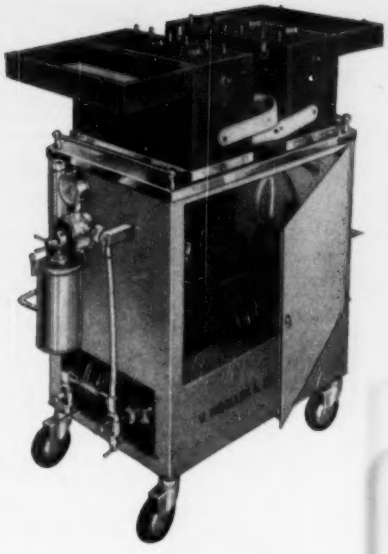

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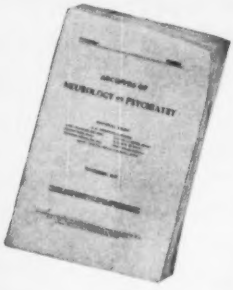

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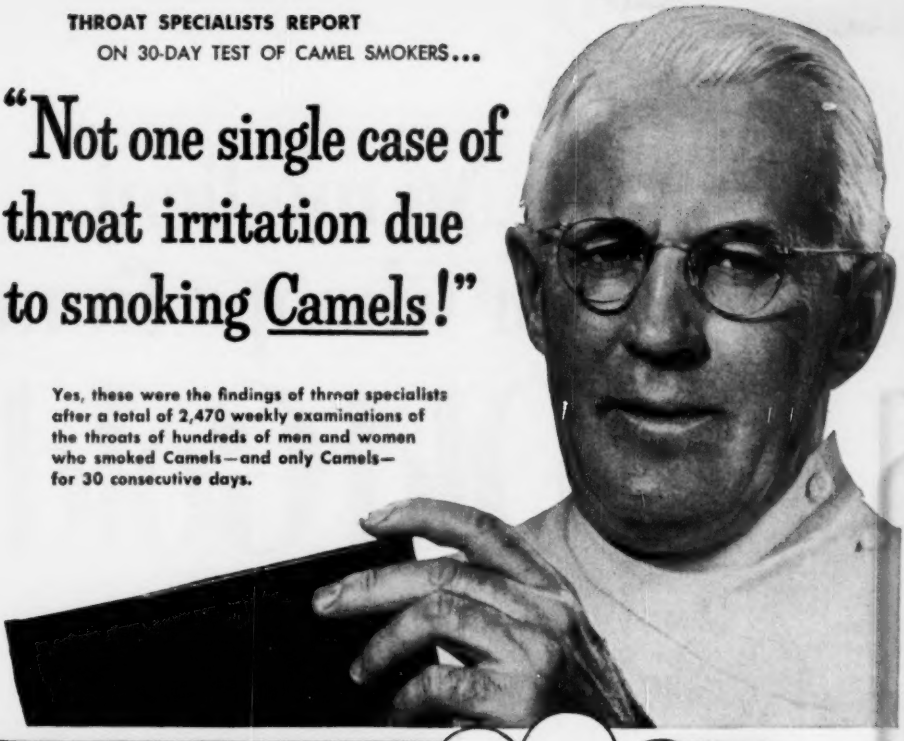

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